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Original Articles

MAXILLARY SINUSITIS OF DENTAL ORIGIN

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IT HAS been generally held that the frequency of inflammatory processes of the maxillary sinus of dental origin is due to the proximal anatomical relationship of the maxillary teeth to the maxillary sinus. However, there is still no agreement as to the ratio between the sinusitis of a dental origin and that of a nondental origin. The dentists emphasize the overwhelming importance of dento-alveolar abscesses to the involvement of the maxillary sinus, while the rhinologists point to infectious or allergic diseases and to nasal diseases as main factors of inflammation of the sinus mucosa. Clinical observations and mainly x-ray findings are the basis upon which these divergent conclusions are founded. Although these practical means are of extreme importance as the only means available to the practitioner, I consider the microscopic examination of human maxillae obtained by chance at autopsy as being more helpful in shedding light upon our problem. By reporting on such microscopic study, the present paper serves the purpose of widening our point of view regarding the dental origin of sinus diseases and of showing that even a thick bony wall between the maxillary sinus and diseased teeth, roentgenographically and microscopically evidenced, does not exclude a dental origin. Furthermore, this investigation will prove that the sinus mucosa may be affected even by a so-called "pyorrhea" condition or even by a chronic pulpitis of a neighboring tooth.

One of the greatest authorities among the rhinologists, M. Hajek,[1] mentioned this possibility in his textbook and merely theorized on it. He specifically stated: "The theory of the invasion of the alveolar process through infection of a root of a tooth has already been advanced and accepted by different authors. The absence of a communication between the alveolus and the maxillary antrum, after extraction of a tooth in an empyema of clinically probable dental origin, seemed to prove conclusively the extension of the diseased process through the bone." Hajek[1] continued, "To me it is very probable that infection may extend from the tooth alveolus through the bone as already proved in the posterior plate of the frontal sinus and the roof of the sphenoid sinus. In this case there is no visible macroscopic change, but the microscopic examination may explain the path of infection. . . . It would be a very fortunate incident if the anatomists should obtain such a fresh case suited for examination at the post-mortem table. . . . We are not able to prove this in the living. After the disclosure of this anatomico-pathologic possibility the frequency of dental empyema cannot be accurately estimated."

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Read before a combined session of the St. Louis Dental Society, Radiologists, and Otolaryngologists of St. Louis, Mo.

The following report on the microscopic findings of sixteen human specimens removed at autopsy will evidence this possibility which Hajek considered and will compose a more complete picture of the dental-antrum relationship. All of the specimens were embedded in celloidin and were mostly cut in a buccolingual direction.

Being fully aware of arguments that might arise as to the value of this method of investigation, I included in my study only those cases in which the most evident pathologic changes of the sinus mucosa were confined to the area close to the teeth and the structural changes around these teeth being such that a causal relationship between the findings in the tooth-supporting structure and the sinus involvement could not be doubted. Moreover, instances of a total sinusitis have not been taken into consideration in order to eliminate cases which could be regarded as deriving from another source. Gross anatomic studies are insufficient in establishing the importance of the anatomic relationship between the maxillary sinus and the teeth to the causation of sinusitis. Therefore, I included in my study the microscopic examination of normal maxillae in order to determine the tissue pathways along which a lesion of the tooth or its supporting tissue might extend to the maxillary sinus. A search of the literature on this subject produced only one paper, written by A. Strubell,[2] in which the author discussed the intimate relationship between the blood vessels supplying the teeth and their periodontal tissue and the sinus mucosa. Strubell injected the vessels of human maxillae with dyes and dissected them in sections of 20μ to 50μ. He stated that the blood supply of the maxillary sinus mucosa, the spongy bone of the maxilla, the periodontal tissue and the pulps of the adjacent teeth is provided by a common system of blood vessels which communicate by dense anastomoses with each other. There is no doubt that infectious processes might easily proceed from the tooth alveoli along this extended network of capillaries into the maxillary sinus in spite of a thick bony wall between the tooth and the antrum. It is interesting to note that there is no correlation between the findings of Strubell and the studies of lymphatics of nasal sinuses by André.[3] André, in direct contradiction to Strubell's statement, could not demonstrate any connection between the dense network of lymphatics of the maxillary sinus and that of the supporting tooth tissue. This important anatomic subject should be restudied.

My microscopic findings in normal maxillae show that the bony floor of the antrum is not always continuous; that is, it is not only pierced by vessels at intervals, but also occasionally shows broad interruptions so that the apical periodontal membrane of the neighboring teeth becomes directly adjacent to the mucosa of the sinus. Thus, my findings supplement the very valuable study of A. Strubell. Figs. 1 and 2 demonstrate the afore-mentioned broken bony antrum floor. Microscopically, the apical areas of the intact periodontal membrane were found to be directly covered by the antral mucosa. The bone trabeculae bordering these gaps did not reveal any pathologic changes although the intertrabecular marrow had undergone a fibrous transformation. However, there was no evidence of inflammation anywhere in the vicinity.

The variability of the histologic structure of the normal antral mucosa must be emphasized. Its structure varies not only between other maxillary

sinuses but is not uniform even within one individual sinus. The sinus mucosa of the nasal sinus wall resembles that of the nasal mucosa. It is somewhat looser, yet it is composed as is the nose mucosa of a pseudostratified, ciliated columnar epithelium and an underlying lamina propria with a structureless basement membrane. It contains lymphatic tissue, mixed mucous glands, a periosteal, glandular, and a subepithelial system of capillaries, and a dense network of lymphatics. The fibrous layer of the lamina propria immediately adjacent to the bony wall is practically the periosteum. In contrast to this part the mucosa of the floor and the distal parts of the maxillary sinus is occasionally much thinner. The epithelium here consists of only one or two rows, the

Fig. 1.

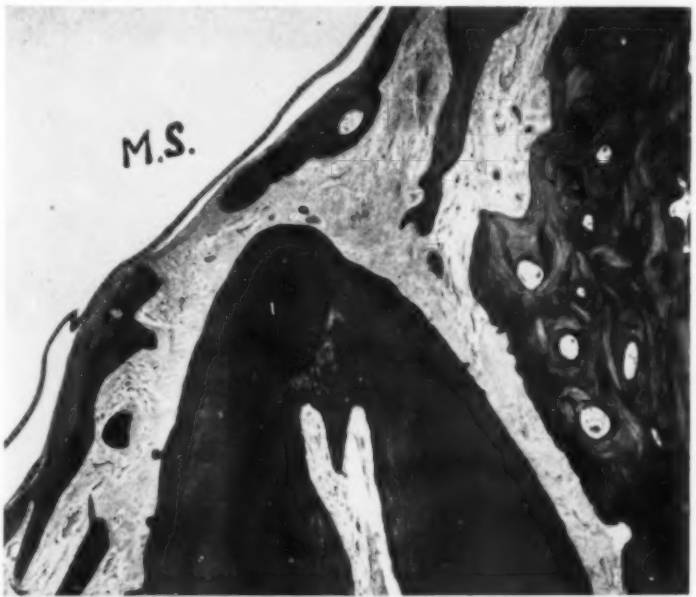

Fig. 2.

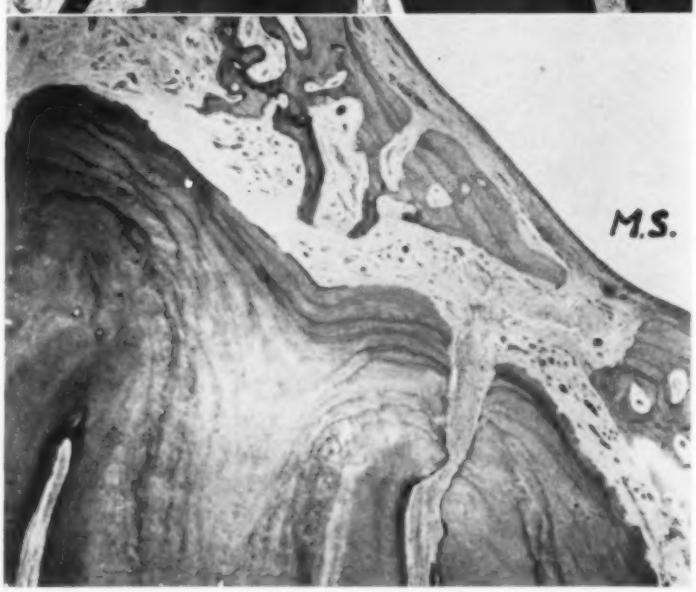

Fig. 1.—Photomicrograph showing an interruption of the bony floor of the maxillary sinus (*M.S.*) through which the intact periodontal membrane communicates directly with the normal mucous membrane.

Fig. 2.—Photomicrograph showing the intact periodontal membrane contacting the mucous membrane of the maxillary sinus (*M.S.*) through an opening in the bony floor.

lamina propria may be missing, and mixed mucous glands may or may not be present (Figs. 3 and 4). There are always lymphocytes and leucocytes either scattered or densely accumulated in the fibrous portion. Larger amounts of these cells, of course, are to be regarded as inflammatory products.

On the basis of these anatomic facts I want to approach the discussion of the possible effect of inflammatory changes of the tooth-supporting structures on the sinus mucosa.

Fig. 3.

Fig. 4.

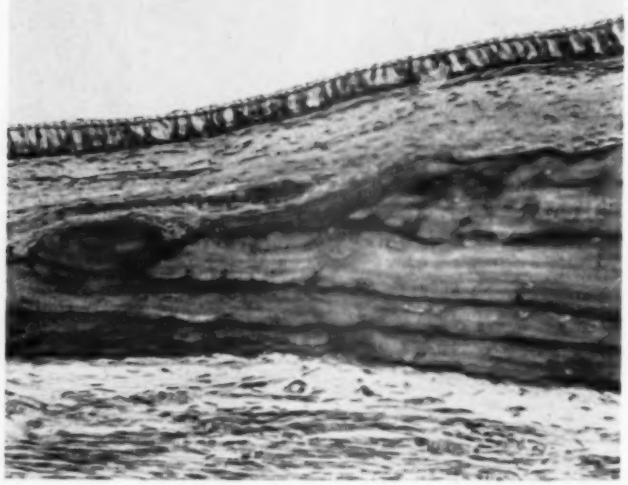

Fig. 3.—Photomicrograph showing the structure of a normal mucous membrane of the nasal wall of the maxillary sinus. Pseudostratified, ciliated columnar epithelium. Structureless basement membrane. Lamina propria with lymphatic tissue, mixed mucous glands and capillaries.

Fig. 4.—Photomicrograph showing the structure of a normal mucous membrane of the floor of the maxillary sinus. The tissue is thinner and more fibrous than that in Fig. 3.

In presenting the description of the microscopic changes of the tooth-supporting tissue and their effects on the antral mucosa, I shall follow this outline:

1. Periapical infection separated by the original thick bone from the maxillary sinus.

2. Periapical infection with perforation into the maxillary sinus reclosed by new bone.

3. Profound pyorrhea (paradentitis profunda) and maxillary sinus.
4. Chronic pulpitis and maxillary sinus.

1. PERIAPICAL INFECTION SEPARATED BY THE ORIGINAL THICK BONE FROM THE MAXILLARY SINUS

Among these lesions the chronic periapical infection of certain upper teeth and its exacerbation occupy the first place. However, if the roentgenogram of the suspected teeth reveals a fairly well-bordered area of periapical bone resorption isolated by a bony layer from the maxillary sinus, and no definite clinical symptoms are found, an involvement of the maxillary sinus of dental origin is usually not considered. The reason for this is that the term "granuloma" generally and indiscriminately used for all types of periapical inflammation which may be revealed by roentgenograms developed the conception that a periapical infection is a granulation-tissue area encased by a fibrous membrane that definitely acts as a barrier against involvement of the surrounding tissue. This erroneous idea must be discarded since the microscopic studies of many human specimens by Häupl and Bauer[4] proved that, in spite of a connective tissue membrane, bone marrow distant from this membrane may show clear evidence of inflammation with more or less noticeable effects on the surrounding bone trabeculae. Microscopically, the periapical infection always appears as a more or less localized osteomyelitis of benign nature which started as a diffuse acute inflammation and assumed gradually the appearance of a chronic proliferative inflammation with formation of fibrous tissue.

The acute infection usually deriving from infected material in the pulp cavity turns the periapical region into a heavily inflamed area with hyperemic blood vessels and exudation of neutrophiles and fluid with rapid destruction of the surrounding bone trabeculae by osteoclasts. It is obvious that an acute process, of this kind, of certain upper teeth usually would not involve the antrum unless there are conditions as demonstrated in Figs. 1 and 2. However, if the prolongation of this inflammatory stage or, as is usually the case, the low pathogenicity of the microorganisms establishes a chronic inflammation, the infection spreads into the more distant bone marrow closer to the maxillary sinus following the path of blood vessels and lymphatics. While the roentgenogram of such a case shows resorption of bone confined only to the periapical region, the microscopic examination of similar instances of my specimens revealed the progress of the lymphocytic infiltration along the vessels of the bone marrow toward the antrum with a subsequent involvement of its mucous membrane restricted to the area next to the bone marrow affected.

Fig. 5 shows a buccolingual section through the apex of the palatal root of a first upper molar and the floor of the antrum, the mucosa of which manifested a cystic growth that protruded into the sinus cavity. The apical end of the root, which was filled with a gangrenous mass, had been resorbed and covered by a thick layer of proliferated epithelium that derived from Malassez's epithelial rests. It was surrounded by dense fibrous tissue and contained a heavy lymphocytic and especially plasma cell infiltration along capillaries filling in interstitial spaces. Although the density of this fibrous structure had increased peripherally and was accompanied by a loss of cells there were many dilated capillaries in this part with heavy perivascular lymphocytic infiltration

which pierced the tissue in various directions and extended in the bone marrow above. All of the sections obtained from this specimen revealed intact bone trabeculae of old structure the surfaces of which were partly coated by small osteoblasts some of which were at rest but no osteoclasts were seen. The absence of any resorption lines within the bone trabeculae evidenced that no resorption prior to the time of the investigation had occurred either. These intact bone trabeculae above the crest of the periapical area encased bone marrow which had been transformed from a fatty marrow into a complete fibrous or a partially fibrous marrow (Fig. 6). This transformed marrow contained dilated and thrombosed veins with perivascular cell infiltration and exudate. These changes of the bone marrow restricted to a narrow area of the bone which separated the periapical infection from the maxillary sinus indicated the path of the infection toward the antral mucosa. The inflammation was clearly confined to the floor of the mucosa.

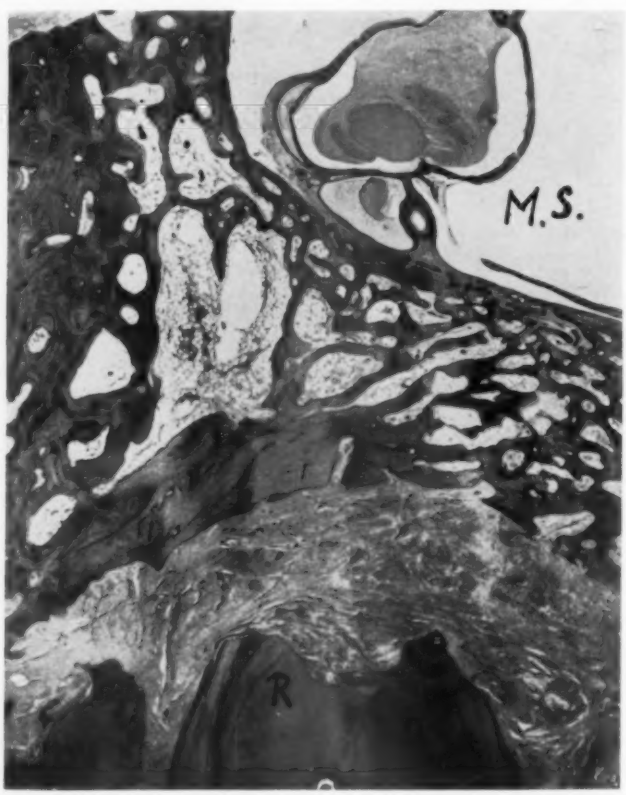

Fig. 5.—Photomicrograph showing the chronic periapical inflammation of the resorbed palatal root (*R*) of a first molar separated from the maxillary sinus (*M.S.*) by thick bone. Cyst formation of the chronically inflamed mucous membrane of the sinus.

The mucous membrane was found to be very vascular (Fig. 7). It contained dilated vessels occasionally thrombosed. Scattered throughout the fibrous ground substance there were massive lymphocytic infiltrations around the vessels and dilated mucous glands with their fluid secreted. The small cell infiltration was particularly excessive in the subepithelial tissue. Small abscesses were observed here and there. There were areas of collected exudate around some of the larger vessels. The epithelial lining of the mucosa showed

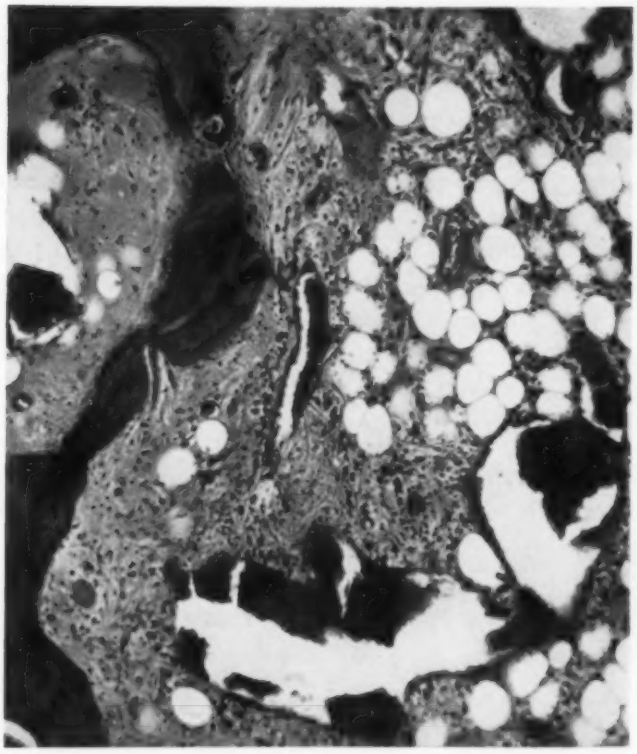

Fig. 6.—Photomicrograph (higher magnification) showing bone marrow of the thick bony layer between apical lesion and maxillary sinus (Fig. 5). Dilated capillaries, thrombosed veins, exudate, perivascular infiltration of fibrous tissue replacing fat marrow thus demonstrating the path of the infection into the antrum.

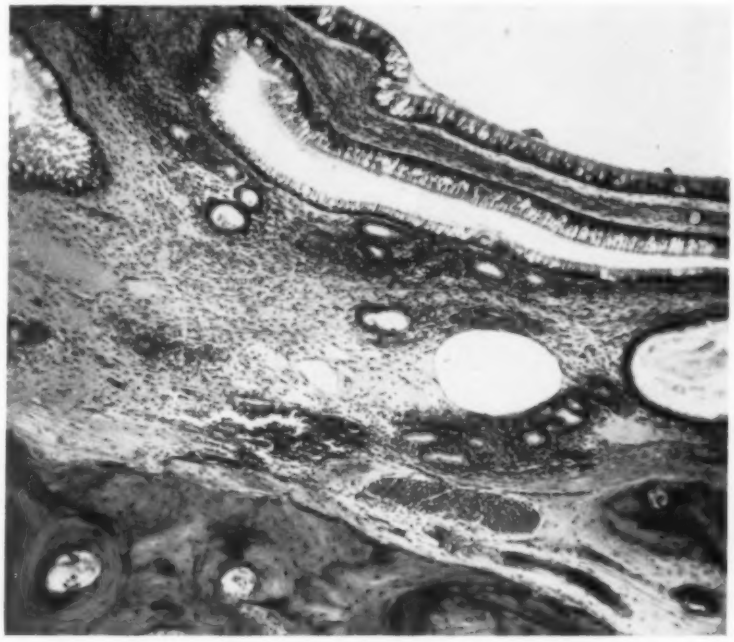

Fig. 7.—Photomicrograph (high magnification) showing the chronically inflamed antrum mucosa of Fig. 5.

an abnormally large number of goblet cells and was partially covered by fibrin. The epithelial layer of the mucous membrane covering many papillary formations, which projected into the cavity and which were produced by the proliferation of the epithelium, was hypertrophic. Just above the periapical infection, cysts of various sizes bulged into the sinus. Some of them were lined with pseudostratified epithelium, others, the larger ones, with a single row of flattened epithelial cells. Microscopically, the mucosa of the part in question revealed a picture of a chronic inflammation which resulted from the apical infection and was transmitted via the blood vessels of the bone marrow through a thick bony wall.

Another type of inflammatory change of the mucous membrane of the maxillary antrum transmitted from a chronic periapical abscess through an intact bony floor of the antrum is shown in Figs. 8 and 9.

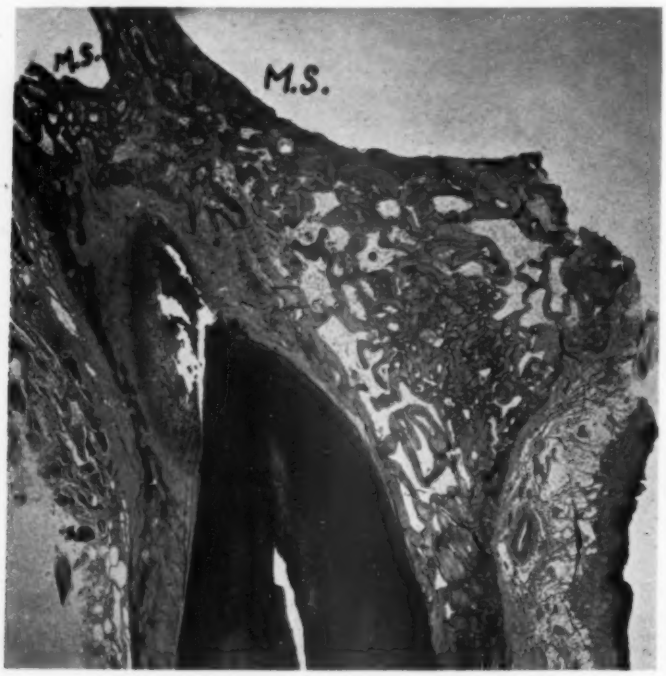

Fig. 8.—Photomicrograph of an upper second premolar with periapical abscess which is separated by thick bone from maxillary sinus (*M.S.*).

A large area of the alveolar fundus of the root of an upper second premolar was resorbed and replaced by a granulation tissue that surrounded an abscess located laterally to the apex. Although some trabeculae of the bone separating the apical process from the antrum have been destroyed and were rebuilt in part, the bony floor had not yet been affected. However, the infection evidently extended along the bone marrow to the floor and from here along the perforating veins into the mucous membrane. The mucosa of the floor alone was involved. Microscopically, the latter showed extreme edematous changes occupying mainly the medial and subepithelial part of the thickness of the mucosa which was more than twice as thick as normal. In the area of the mucous membrane adjacent to the apical infection this extraordinary accumulation of edema produced pseudocysts of considerable size.

Edema was also very evident in the periosteal layer of the mucosa. Microscopically, this limited part of the mucosa showed a chronic catarrhal inflammation derived from a considerably distant apical tooth lesion. There were dilated vessels in the loose tissue surrounded by a moderate amount of lymphocytes and plasma cells. Large hemorrhages were scattered throughout the mucous membrane which was covered by a multilayered epithelium. Mucous membrane changes became less distinct in the regions farther away from the periapical lesion.

My experience with this type of involvement of the mucosa of the maxillary antrum covers nine cases of a total of sixteen collected and studied, thus possibly indicating the more common extension of a periapical tooth lesion into the maxillary antrum.

Fig. 9.—Photomicrograph (higher magnification) showing the very edematous thickened mucous membrane of the maxillary sinus of Fig. 8., with perivascular lymphocytic infiltration and hyperplastic epithelial layer.

2. PERIAPICAL INFECTION WITH PERFORATION INTO THE MAXILLARY ANTRUM RECLOSED BY NEW BONE

The perforation of a periapical process into the maxillary antrum has been held to be the common cause of a sinusitis so far. Microscopic studies related to this genesis were conducted by Thoma,[5] Kronfeld,[6] Hoepfel,[7] and others. The microscopic appearance of such cases as one would expect is in accordance with the roentgenographic findings which reveal a more or less wide opening of the bony antrum floor in direct connection with an apical tooth lesion. Cases of this sort are well known and the present paper will consider this type only wherein the microscopic examination showed a reclosure of the perforation by layers of new bone. As these newly deposited bone trabeculae which are a reactive product of chronic inflammation tend to calcify very readily, they appear in the roentgenogram falsely as an intact bony floor. Roentgenographic examination of such cases is insufficient evidence for proving the inflammatory gene-

sis of this new bone tissue, whereas, on the other hand, microscopic examination left little doubt as to such a genesis. The connective tissue reaction as the fundamental part of chronic inflammation tends to repair the damage by producing new bone. These bone trabeculae laid down to overbridge the gap in the antrum floor were irregularly arranged and their cells were large (Figs. 10 to 14). Many of these trabeculae coated by a dense layer of osteoblasts were still in the osteoid stage of bone development and very cellular (Figs. 12 and 14). Others being somewhat older became hypercalcified due to the chronic inflammation. The microscopic examination clearly showed that the perforation was reclosed by a new bone partition, which divided the whole chronically inflamed area into two parts, both containing infected granulation tissue (Figs. 11, 12, and 14). Figs. 10, 11, and 12 represent one of these cases. The bone marrow in the immediate and even more distant vicinity of the periapical lesions of these cases was converted into a dense fibrous granulation tissue with hyperemic and thrombosed vessels and the old neighboring bone trabeculae were covered by osteoblasts. There was a very noticeable round cell infiltration surrounding the blood vessels and also interspersed within the connective tissue.

Fig. 10.—Photomicrograph showing a second premolar with an extended chronic periapical infection separated from the maxillary sinus (*M.S.*) by a thin layer of newly deposited bone.

The surface of the old bony floor toward the maxillary sinus was scalloped by Howship's lacunae containing fairly large osteoclasts. However, the newly formed bony bridge aforementioned was lined with osteoblasts.

The antral mucosa (Figs. 11 and 12) was transformed into granulation tissue and its epithelial lining had almost entirely been destroyed. Widespread hemorrhages within this tissue adjacent to the bony floor were observed while extravasation occupied the subepithelial layer.

Another instance of this group is pictured in Figs. 13 and 14. A very extended periapical abscess around the root of a second upper premolar was surrounded by a distinct pyogenic membrane and a broad layer of dense fibrous tissue concentrically arranged. Due to an acute exacerbation the bony floor of the maxillary sinus has been perforated thus involving the mucous membrane. This opening was found to be obliterated by easily recognizable new bone formation which occasionally assumed a remarkable thickness.

Fig. 11.

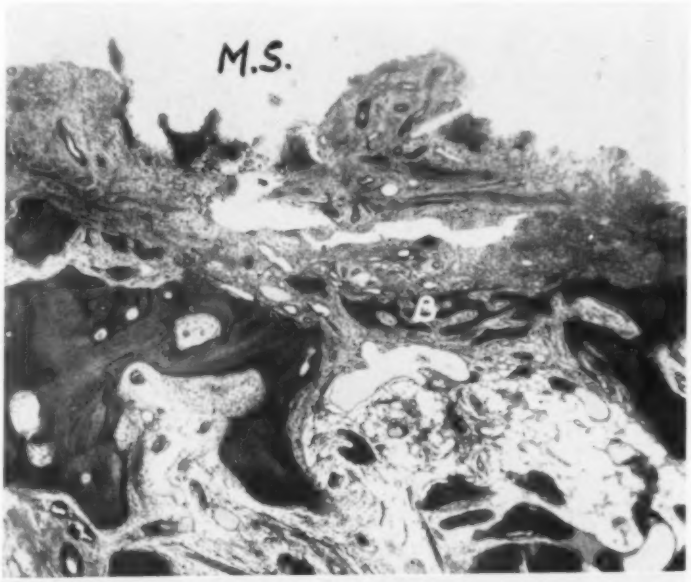

Fig. 12.

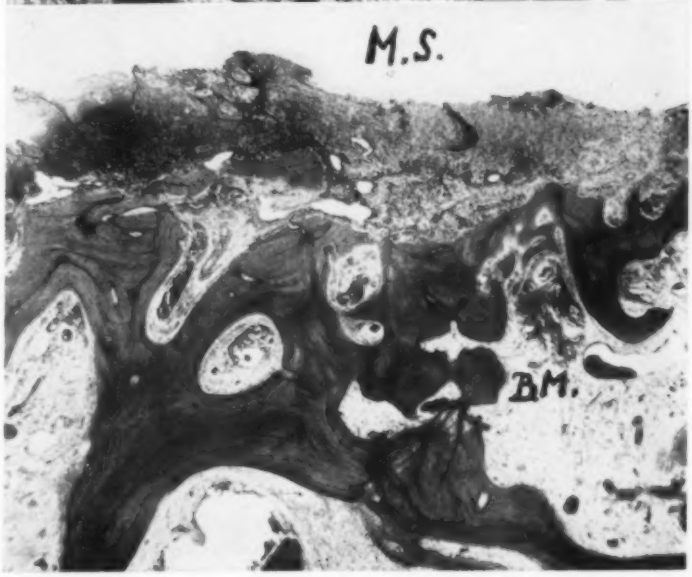

Fig. 11.—Photomicrograph (higher magnification) of Fig. 10, showing newly deposited bone trabeculae (*B*) between the inflamed bone marrow and the mucous membrane of the maxillary sinus (*M.S.*) which is transformed into a granulation tissue with extensive lymphocytic infiltration.

Fig. 12.—Photomicrograph showing newly formed bone trabeculae separating bone marrow (*B.M.*) with dense perivascular infiltration from heavily inflamed antrum mucosa the epithelial lining of which was destroyed.

The severe involvement of the antral mucosa was clearly restricted to this area and decreased gradually in its intensity toward the periphery. The

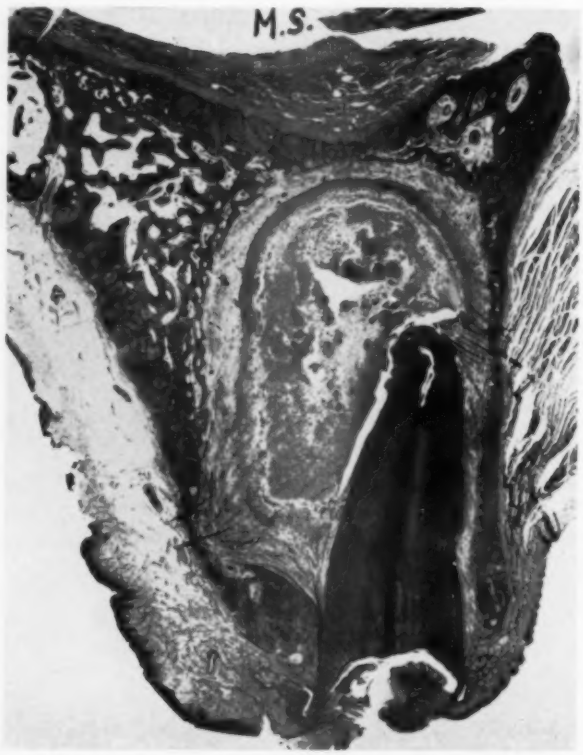

Fig. 13.—Photomicrograph showing the root of an upper second premolar with a very extended abscess surrounded by a pyogenic membrane and a dense fibrous layer. The perforation into the antrum (*M.S.*) has been closed by new bone trabeculae.

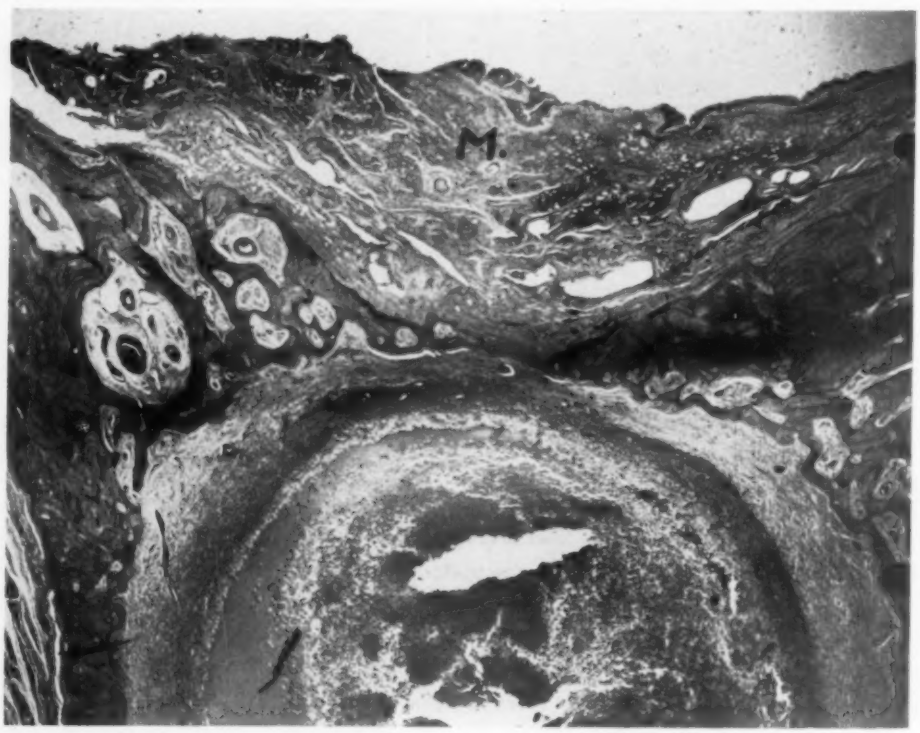

Fig. 14.—Photomicrograph (higher magnification) showing the closed perforation into the antrum of Fig. 13. Severe involvement of the antrum mucosa (*M.*).

mucosa presented an intense chronic inflammation with pronounced vascularity, perivascular and periglandular round cell infiltration, an abundance of mucous glands, and a very definite hyalinization of the connective tissue. The latter change was predominately seen in the subepithelial layer of the region affected and bordered by an unusually densely compacted round cell infiltration. The epithelial layer composed of flattened cells was partly hypertrophic and partly broken down. Occasionally, epithelial proliferation into the underlying tissue was observed.

Fig. 15.

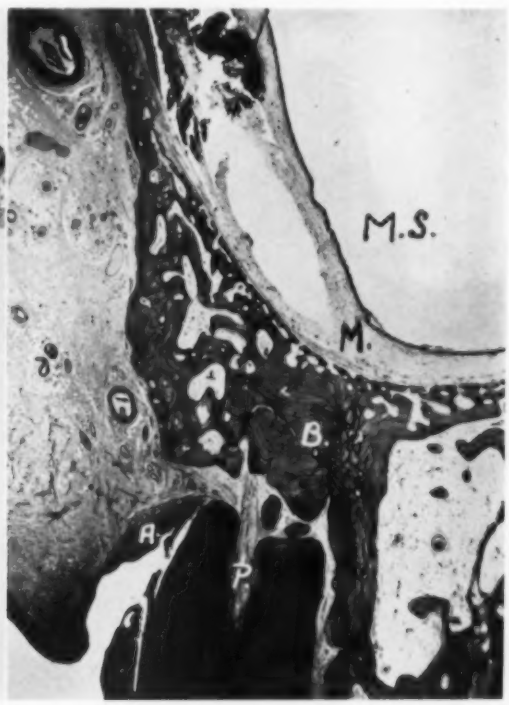

Fig. 16.

Fig. 15.—Photomicrograph (buccopalatal section) showing the involvement of the mucosa of the maxillary sinus (*M.S.*) from a profound "pyorrhea" (paradentitis profunda) of a first molar. Inflammation traveled through the bone marrow into the antrum mucosa.

Fig. 16.—Photomicrograph (higher magnification) showing the apical third of the palatal root of Fig. 15. Epithelial attachment (*A*) on the apex with dense cell infiltration, fibrous pulp tissue (*P*), resorption of the apical surface repaired by bone-cementum, very dense bone (*B*) with numerous incremental lines above the root end thus indicating rhythmic apposition. Edema and large hemorrhages in the mucosa (*M*).

3. PROFOUND PYORRHEA (PARADENTITIS PROFUNDA) AND MAXILLARY SINUS

The specimens examined included three instances of involvement of the mucosa of the maxillary sinus resulting from a so-called advanced pyorrhea. The microscopic examination of the pulps did not reveal any pathologic changes that could have produced the antrum lesions. The pulps of the teeth shown in Figs. 15, 16, 17, 18 contained masses of secondary dentine fused together with denticles and dystrophic calcifications as well and were transformed into a rather dense scarlike fibrous tissue. Some sections of the tooth shown in Fig. 18 demonstrated fairly well-preserved odontoblasts covering normally wide zones of dentinoid while others revealed a reticular atrophy of the pulp tissue with vacuolization of the odontoblasts.

All these pulp changes could easily be reconciled with the very advanced "pyorrhea" in which the bottom of the pockets extended to the apical foramina (Figs. 15, 16, and 17) or exposed the entire roots (Fig. 18). The epithelial lining of the deep crevices shown in Figs. 15, 16, and 17 was thickened or was ulcerated in Fig. 18, due to the severe inflammation that had prepared the path

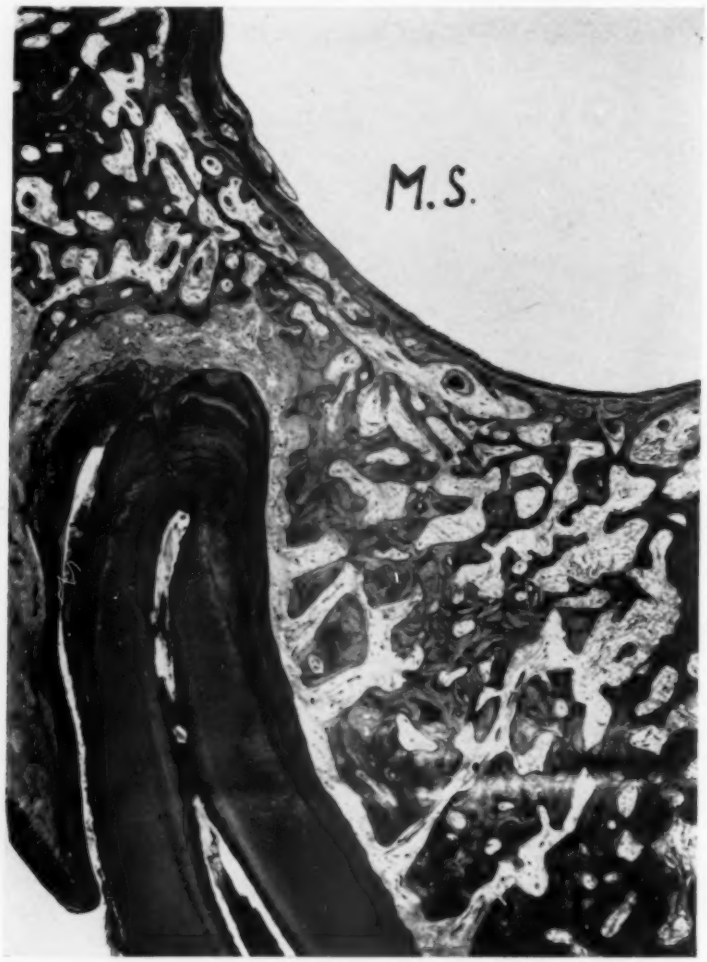

Fig. 17.—Photomicrograph (medium magnification) showing a part of the palatal root of a second molar. Gingival pocket reaching to the apical foramen, the epithelial lining accompanied by very dense lymphocytic infiltration. Chronic inflammation traveling through the bone marrow toward the antrum. The mucosa shows typical chronic inflammation restricted to the area in the vicinity of the root end.

for the migration of the epithelium along the cementum and was surrounded by a very dense infiltration of lymphocytes and plasma cells. These round cell accumulations extended through the apical part of the periodontal membrane into the bone marrow beneath the antrum floor. The fairly well-preserved periodontal structure, although thickened (Figs. 15, 16, and 17) was converted in the most advanced case (Fig. 18) into an edematous granulation tissue. The original anatomic relationship between the root ends of the first molar and the maxillary sinus seen in Fig. 15 must have been a very intimate one as the changes of the buccal part and its alveolar fundus indicated. The interpretation of Fig. 16 revealed that this root end was originally closely related to the antrum floor.

However the resorption of the root end as the result of the advancing pyorrhea and the tipping of the tooth toward the palate resulted in the deposition of an almost solid block of bone tissue between the root end and the antrum with numerous parallel incremental lines indicating a rhythmical deposition of bone (Fig. 16). In addition, there was a layer of newly formed bone trabeculae deposited by the periosteum of the antral mucosa upon the original bony floor.

Fig. 18.—Photomicrograph (low magnification) showing the elimination of an upper second molar from its socket due to profound pyorrhea. Periodontal membrane transformed into granulation tissue. Chronic inflammation invaded the antrum mucosa through the bone marrow.

The bone marrow of these cases in question was converted into fibrous tissue and round cells, principally lymphocytes, and plasma cells were interspersed between the dilated vessels. These changes affected only the bone marrow above the root ends. It was due to these extensions into the marrow that the mucosa of the maxillary sinuses was affected. It must be noted that the involvement of the mucous membranes was distinctly limited to areas close to the root ends.

The mucous membranes of these cases showed a great variety of alterations. The mucosa in Fig. 15 was very edematous and contained large hemorrhages

that extended from the periosteal zone into the subepithelial layer occasionally occupying the whole thickness. The epithelial lining containing many goblet cells was hyperplastic and an intense lymphocytic infiltration was seen in the subepithelial layer. The mucosa in Fig. 17 affected in the same way revealed a different picture. Cyst formation and epithelial proliferations into the mucosa forming papillary elevations stood out clearly. The perivascular and periglandular tissue was densely infiltrated with lymphocytes and plasma cells which accumulated beneath the multilayered epithelium. The most striking feature, indeed, was observed in the third case (Fig. 18), the mucosa of which was deprived of its epithelium in the area affected. Necrotic parts of it had sloughed off and closely packed masses of lymphocytes filled the entire thickness of the membrane (Fig. 19). Thrombosed vessels and pseudocysts with exudate were found in the mucosa.

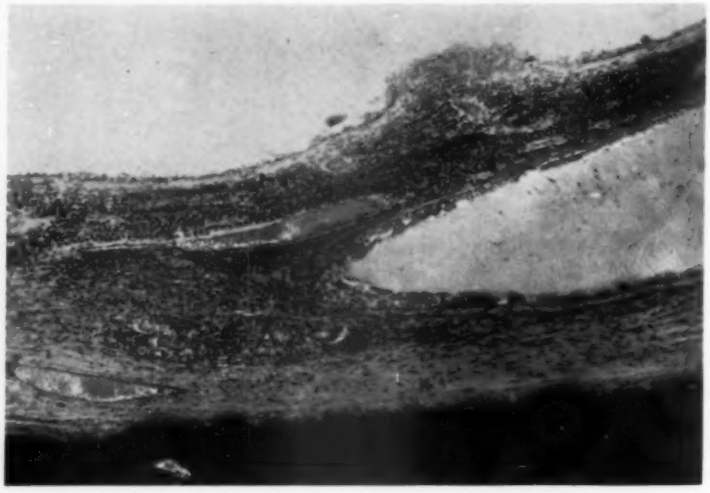

Fig. 19.—Photomicrograph (higher magnification) showing the mucous membrane of the maxillary antrum of Fig. 18. Very dense lymphocytic infiltration, destruction of the epithelial layer, pseudocyst formation.

Of course, it might be argued that the involvement of the sinus mucosa in these cases developed from another source, independent of the teeth in question. However, the fact that the lesions of the mucosa were restricted to the alveolar sinuses above and that the path of infection from the periodontal tissue into the sinus stood out clearly supports my conception that the lesions of the maxillary sinus resulted from the profound paradentitis (pyorrhea).

4. CHRONIC PULPITIS AND MAXILLARY SINUS

This section will deal with the local changes I observed in the mucous membrane of the maxillary sinus as a result of a case of chronic pulpitis. One might be amazed that a pulp which was partially converted into granulation tissue, but still containing living parts, might affect the antrum even without producing an apical infection. Such a condition can develop only if the tooth is in close anatomic relationship to the antral mucosa and the infection of the pulp is mild but of long standing. Furthermore, the assumption of a particular susceptibility of the mucosa due to prior attacks from other sources must not be disregarded.

Here again microscopic evidence points to the fact that the network of blood vessels and lymphatics plays the main role of transmitting the infection to the mucous membrane. One of the specimens I obtained by chance and studied microscopically demonstrated strikingly and convincingly the advance of the infection from a living but partially affected pulp toward the antrum. The pulp of a second premolar exhibited a low-grade chronic inflammatory lesion confined to the coronal part, and a definite, dense perivascular infiltration with lymphocytes took the path along the alveolar nerve to the floor of the maxillary sinus (Fig. 20). In spite of a small amount of edema that followed the course of the nerve and the vessels above the apical foramen

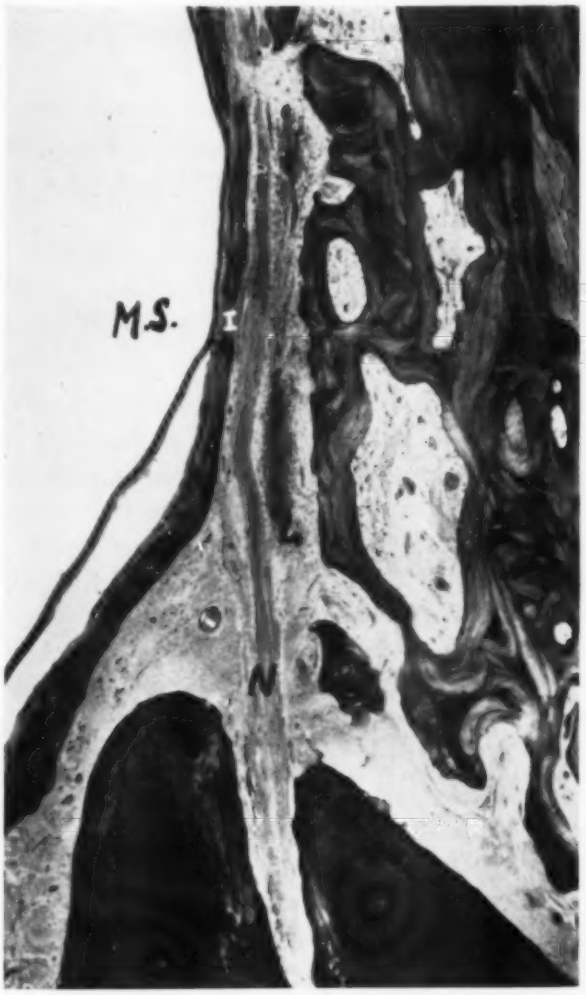

Fig. 20.—Photomicrograph (medium magnification). Perivascular lymphocytic infiltration (*L*) following the dental nerve (*N*) and deriving from a partial chronic pulpitis. Note the unaltered periapical area and the interruption (*I*) of the bony antrum (*M.S.*) floor through which the infection reached the mucous membrane.

there was no microscopic evidence of any resorption of the bone trabeculae. The adjacent bone marrow, of course, had changed into fibrous tissue with little inflammatory exudate. It was the narrow interruption of the bony antrum floor through which the infection had been introduced into the mucous membrane. However, there was no material reaction on the part of the mucous

membrane. This interesting instance is very remarkable in that it is the first demonstration that an infection may actually be carried from a partial chronic pulpitis toward the antrum.

SUMMARY

On the basis of microscopic examination of sixteen specimens obtained at autopsy it appears that the roentgenographic picture is not always reliable enough to prove or disprove the dental origin of an involvement of the mucous membrane of the maxillary sinus. There is no difficulty in reconciling the changes of the antral mucosa with a periapical lesion that has perforated the bony floor of the maxillary sinus. However, the findings of my studies seem to indicate that the mucosa of the maxillary sinus is often affected by periapical lesions which are separated from the floor of the antrum by a bony wall of even great thickness. It is the network of blood vessels and lymphatics extending from the periodontal membrane into the antral mucosa that forms the path for the spread of the infection through the bone marrow. Perivascular infiltration with lymphocytes and plasma cells proceeds from the apical focus through the bone marrow, which is transformed into fibrous tissue containing thrombosed vessels, into the maxillary sinus, causing an inflammatory reaction of its mucous membrane.

However, these alterations of the membrane which may derive also from a so-called profound "pyorrhea" and even from a chronic pulpitis are more or less limited to the part of the mucous membrane near the dental focus. The changes of the mucous membrane show all gradations of a chronic inflammation involving its periosteal layer, the mucous membrane proper, and the pseudo-stratified ciliated epithelium. The transformation of the antral mucosa into a dense fibrous tissue seemingly represents a process of repair. The present study does not allow the adoption of any conclusions as to the extension of these more or less localized lesions of dental origin but broadens remarkably our point of view on the relationship between teeth and inflammatory diseases of the mucous membrane of the maxillary sinus.

From a clinical point of view it would be important to ascertain whether or not these more or less local lesions of dental origin of the mucous membrane of the maxillary sinus provoke clinical symptoms and whether or not they disappear with the removal of the tooth from which they originate. A solution of this problem can only be achieved by the cooperation of dentists and rhinologists.

Despite the fact that I am a firm believer in the possibility of a successful conservative treatment of infected teeth, and appreciate the advantages of root resections following properly treated pulp canals, I consider extraction of teeth such as described in this paper the only treatment of choice.

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BONE HETEROPLASIA IN A CASE OF DILANTIN HYPERPLASTIC GINGIVITIS

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THIS case of dilantin hyperplastic gingivitis is reported because of the finding of calcified masses in the corium of the gingivae during routine serial sectioning of biopsies of a series of such cases under study. While observations are limited to the gingivae, the presence of such calcifications in connective tissue in other locations is suggested as a possibility.

The patient, L. V., was a single woman, aged 16 years, American of Italian descent.

History.—The patient was admitted to the Neurology Department of the Vanderbilt Clinic in January, 1939, for treatment of convulsions. At that time she gave a history of "fits for the past two years," with treatment consisting of luminal. During the six months prior to admission at Vanderbilt Clinic, the seizures became progressively more severe. They were of almost nightly occurrence and lasted about five minutes. The attacks were characterized by pallor and rigidity. The eyes rolled up. Daytime attacks also occurred. A diagnosis of "idiopathic grand mal" was made.

The patient was placed on phenobarbital (0.03 gram) and dilantin (1.5 grains) twice daily. Epileptic seizures continued, varying from 2 to 5 per week. Eight months later, the patient complained of irregular menses accompanied by cramps, for which 11 c.c. of antuitrin S was injected in a period of two weeks without beneficial effect.

The dilantin dosage was now increased to three capsules daily. For ten days following each menstrual period both dilantin and phenobarbital were taken four times daily. This medication decreased the number of seizures to about three a month, but frequent headaches, constipation, and fatigue persisted. The dosage was stabilized at four dilantin capsules and three phenobarbital pills daily and continued until the present time. Occasional attacks, much less severe in nature, are still experienced.

Oral Examination.—Gross: All teeth are present and in fairly good occlusion. A series of dental x-rays revealed the presence of several small cavities but alveolar bone is normal. Oral hygiene is poor. The alveolar gingivae are hypertrophied, hard and firm, giving an appearance of a generalized fibromatosis. They are a normal pink color similar to the surrounding mucous membranes with the exception of the marginal gingivae which appear congested. These are the usual findings in dilantin hyperplastic gingivitis.

Microscopic: A biopsy of the upper right No. 3 interdental papilla (extending from the crest to the mucobuccal fold), showed a thickened alveolar gingiva with both the epithelial and connective tissue contributing to the enlargement.

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Epithelium: The alveolar epithelium presents a superficial keratin layer beneath which is seen a layer of parakeratosis, two to four cells deep. There are also present the usual proliferative changes of dilantin hyperplastic gingivitis characterized by the greatly increased number of cells in the prickle and basal cell layers with darkly stained hyperchromatic nuclei and the increase in number of mitotic figures. Some hydropic change is seen. The epithelial proliferation is again accentuated by the downgrowth of the epithelial pegs fairly deep into the corium and by their pointing and frequent splitting.

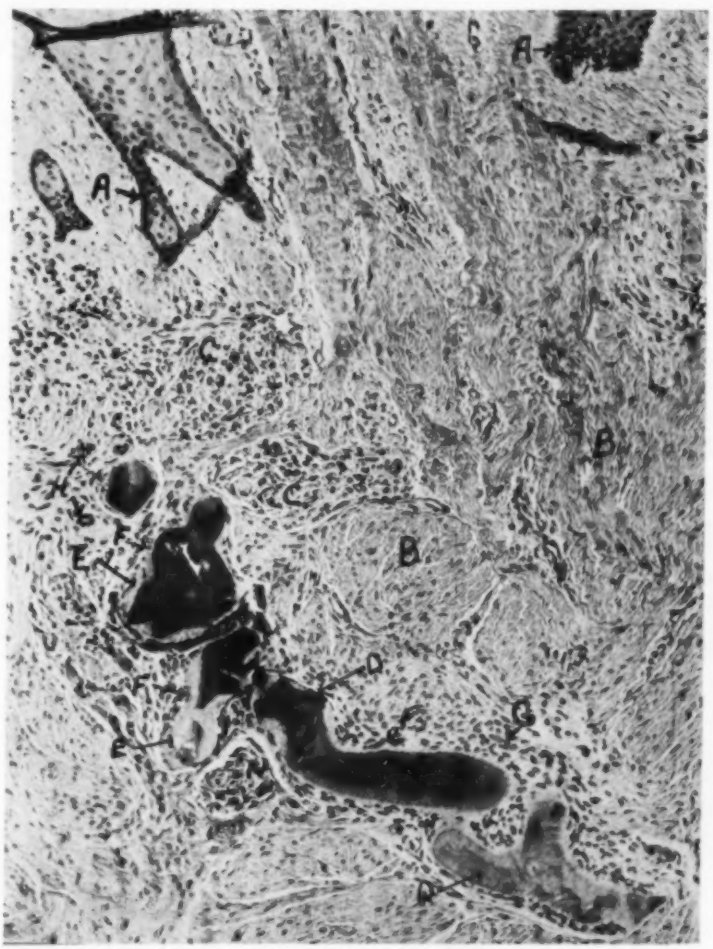

Fig. 1.—Heteroplastic bone in corium of dilantin hyperplastic gingivae. Trichrome stain. Mag. ×100. Alveolar gingiva. *A*, Epithelial pegs. *B*, Dense hyaline bundles of collagen. *C*, Granulation tissue masses rich in capillaries and large fibroblasts. *D*, Heteroplastic bone masses in corium. *E*, Uncalcified organic matrix. *F*, Fibroblasts which have become osteoblastic in function. *G*, Young, active fibroblasts in surrounding areas. *H*, Foreign body giant cells in vicinity of calcified masses.

Connective Tissues: The corium also shows characteristic dilantin hyperplastic changes. The proliferation of connective tissue fibers is prominently seen with thick, dense bundles of collagen forming the basis of the increased size of the corium. Numerous large, young fibroblasts in an apparent state of hyperactivity are seen throughout this layer both scattered in a diffuse pattern and grouped in masses. Inflammatory accumulations are limited to perivascular

arrangements and to the subepithelial region. There is an increase in the size and number of capillaries. Edema is slight. Large nests of granulation tissue are seen in various parts of the corium.

Calcified Masses: Midway between the inner border of the epithelial layer and the periosteal limit of the corium (Fig. 1) are irregularly shaped masses. The central portion of these bodies takes a deep hematoxylin stain, indicating calcium deposition. No lacunae or osteocytes are visible, nor are there any structures simulating Haversian systems. An occasional young connective tissue cell is found in these areas but appears to be accidentally enmeshed in the deposit rather than a normal part of the structure. Surrounding the calcified areas in a narrow zone is an organic matrix which takes a pale eosin stain. Globules of calcospherites are visible along the inner border (Fig. 2). Along the outer border there is an arrangement of irregular rows of numerous mesenchymal cells with large hyperchromatic nuclei similar to the fibroblasts present throughout the corium. These cells appear to be "osteoblastic" in nature and may account for the calcified formations. A small number of foreign body giant cells are irregularly scattered in the surrounding granulomatous areas.

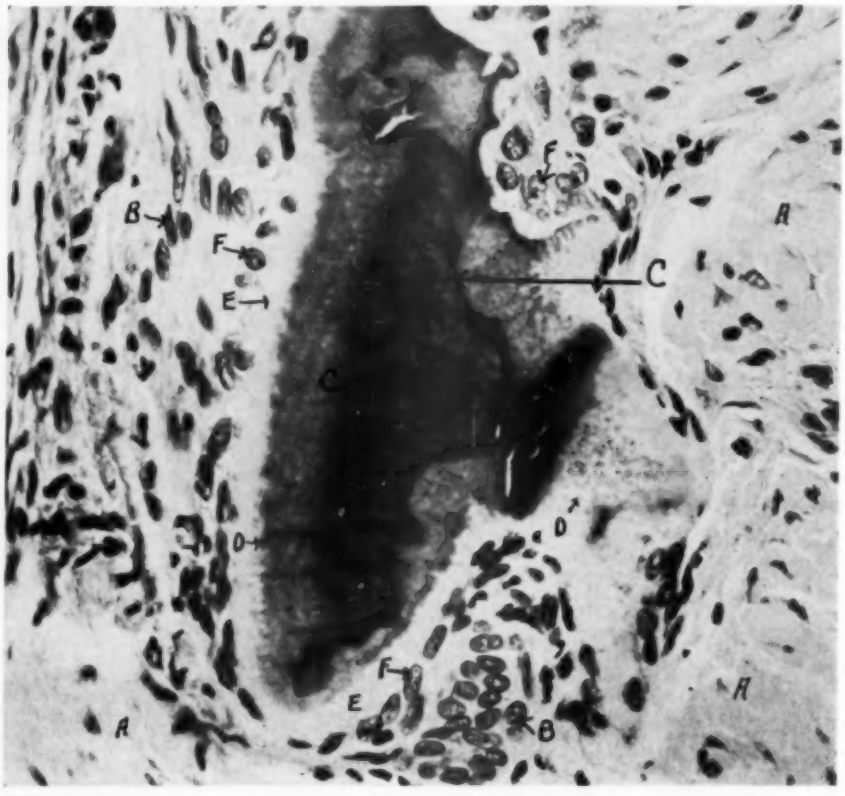

Fig. 2.—High power of a portion of heteroplastic bone in corium. Hematoxylin-eosin stain. Mag. ×420. *A*, Dense collagen. *B*, Active fibroblasts. *C*, Heteroplastic bone. *D*, Calcospherites along outer border of calcified portion. *E*, Uncalcified organic matrix. *F*, Fibroblasts functioning as osteoblasts and similar in appearance to them.

DISCUSSION

Since these calcified masses are completely embedded in the corium of the gingiva and are not in any way connected with the underlying bone, the possibility that they are periosteal extensions may be discarded.

They may be differentiated from true bone formations by the lack of Haversian systems and bone lacunae. Since they possess neither dentinal tubules nor cementocytes in lacunae, they may not be considered either dentine or cementum.

It is generally accepted that stimulated fibroblasts may undergo metaplasia and assume the function of osteoblasts. In these instances the connective tissue stroma becomes hyaline and calcification occurs under the influence of the newly created "osteoblasts."

Because it is apparent that we are dealing with an "unspecialized" hard tissue formation, we may consider the process a bone heteroplasia, a consequence of the metaplasia of dilantin-stimulated fibroblasts acquiring the functions of osteoblasts.

DENTINOMA

Report of Two Cases

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A DENTINOMA is a dental tumor composed chiefly of dentine; it is, by far, the rarest of the hard odontomas.

Thoma reported a case in which a hard spherical mass was situated directly over the occlusal surface of an unerupted, lower first permanent molar. The mass consisted chiefly of dentine, which was covered by a layer of cementum. There were several nutrient canals which extended toward the center of the mass. Straith also reported a case in which he removed a mass from directly over the coronal portion of an unerupted lower third molar. There was no enamel or definite root or crown arrangement in this mass, and the dentine was transversed by canals carrying blood vessels.

REPORT OF CASES

I wish to report two cases of dentinoma that were observed at the Mayo Clinic.

Case 1.—A woman, aged 25 years, complained of discomfort in the molar region of the right mandible. The mucous membrane in this region was red and slightly swollen. It contained an opening which had appeared a short time before the patient came to the clinic. Roentgenographic examination revealed a radiopaque mass situated over and slightly posterior to the crown of an unerupted third molar (Fig. 1). The mass and the tooth were removed with local anesthesia. The dense mass was readily separated from the surface of the tooth and the surrounding bone, and it was removed intact.

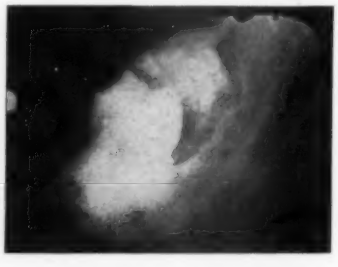

Fig. 1.—Roentgenogram of dentinoma in Case 1.

The mass was decalcified and microscopic sections were prepared. Examination of these sections revealed that the mass was composed chiefly of dentine (Fig. 2) and contained numerous nutrient canals which extended from that part of the growth which had been in close proximity to the crown of the tooth toward the peripheral portion. The surface of the tumor which had been adjacent to the bone of the jaw was covered by a capsule of connective

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tissue (Fig. 3). Although microscopic sections were made from several different parts of the tumor, pulp was rarely observed; however, it was present in one of the sections (Fig. 4).

Fig. 2.—Photomicrograph showing dentine in the dentinoma in Case 1 (×320).

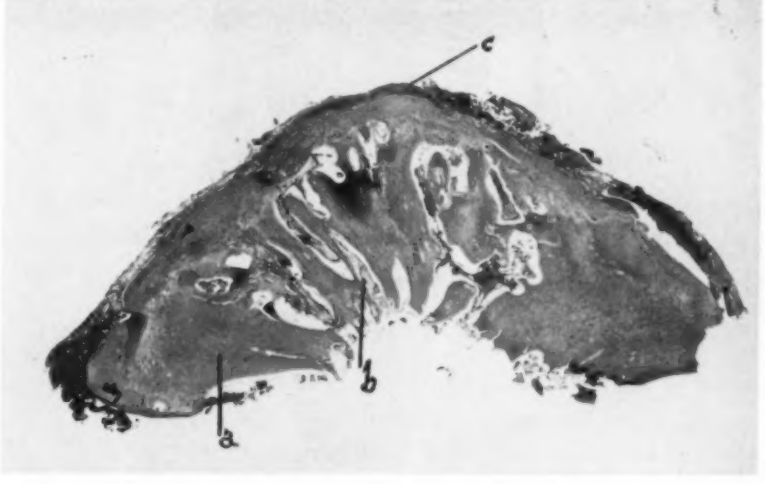

Fig. 3.—Photomicrograph of the dentinoma in Case 1; *a*, dentine; *b*, nutrient canal; *c*, capsule of connective tissue (×10).

Case 2.—The patient was a man, aged 23 years. Roentgenographic examination revealed a radiopaque mass situated directly over the occlusal surface of an unerupted, lower right second molar (Fig. 5). There was no

communication with the oral cavity. When the mass was removed, it could be separated from the tooth and surrounding bone without difficulty. The microscopic appearance of the tumor was similar to that of the tumor in Case 1.

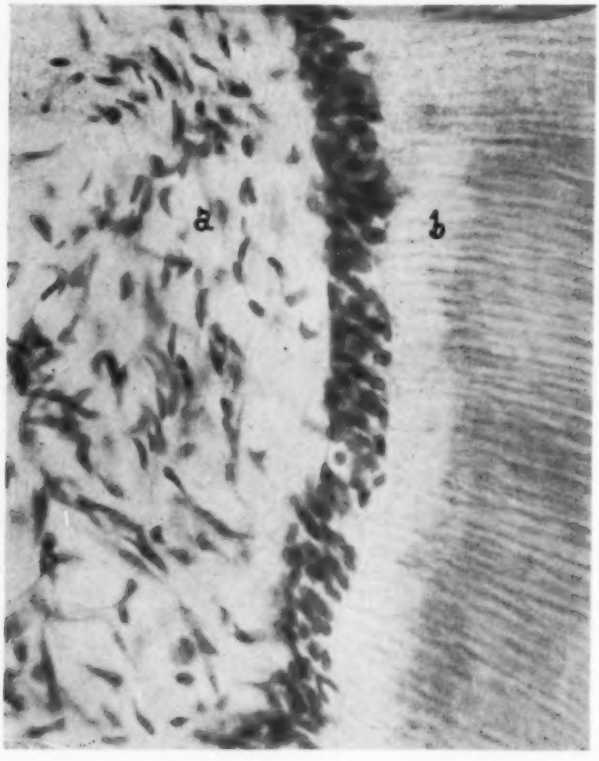

Fig. 4.—Photomicrograph of the dentinoma in Case 1; *a*, pulp; *b*, dentine (×435).

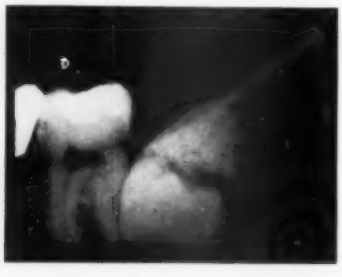

Fig. 5.—Roentgenogram of dentinoma in Case 2.

COMMENT

It is obvious that these two cases and those reported by Straith and Thoma are similar. In all of the cases the dentinoma was situated in the molar region of the lower jaw and was associated with an unerupted tooth. The most striking similarity is perhaps the site in relation to the unerupted teeth—directly over and in close proximity to the coronal portion of the tooth. In the case reported by Straith, the mass fitted over and around the cusps of the unerupted tooth.

It is apparent that the dentine of which these tumors are composed has developed in a region remote from the site of the dental papilla from which

dentine is normally formed. However, its intimate relation to the coronal portion of the unerupted tooth suggests that the dental follicle from which the unerupted tooth developed also may have initiated the development of the tumor.

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AN APPLIANCE FOR USE IN THE CONSERVATIVE TREATMENT OF COLLUM FRACTURES OF THE MANDIBLE, IN MAINTAINING VERTICAL DIMENSION OF THE JAW, AND FOR OVERCOMING SPASM OF THE ELEVATOR MUSCLES OF THE MANDIBLE

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IN A previous communication[1] the authors described a conservative procedure for treating collum fractures of the mandibular condyles based on the application of force exerted vertically downward through the line of the mandibular ramus to overcome the spasm of the elevator muscles of the mandible. Because these muscles insert about the ramus of the bone, a force exerted vertically downward along the line of the ramus is the most effective force in overcoming spasm of these muscles. At first wooden wedges were used to exert the continual vertical force downward along the line of the mandibular ramus.[1] It is readily apparent that such wedges were often difficult to keep in place and could not be used for patients lacking posterior molar teeth. To replace the wooden wedges a simple intraoral appliance was designed which gives satisfactory results on patients who are edentulous, partially edentulous, or have a full complement of teeth. The appliance is adjustable and can be made easily from readily available materials by any oral surgeon.

The appliance is constructed for each patient individually. One form of the appliance used for a patient lacking both upper and lower posterior molar teeth is illustrated in Figs. 1 and 2. A vulcanite or acrylic bite block is made by the usual procedure. Over the level of the third molar area on the side to which the force is to be applied a threaded hole is placed in the bite block. Into this hole is fitted a small threaded jackscrew. The end of the screw near its head is drilled transversely as illustrated (Figs. 1 and 2). By means of a rivet a small metal plate is fastened to the head of the screw so that it rotates freely. This metal plate is padded with sponge rubber to protect the edentulous soft tissues. The appliance is inserted into the mouth with the bite block in place, the jackscrew threaded fully into the bite block, and the rubber-padded metal plate resting against the opposing edentulous ridge (Fig. 1). Care is taken that the jackscrew does not extend entirely through the bite block to prevent impingement on the underlying soft tissues by the end of the screw. Continual force is applied vertically downward along the line of the mandibular ramus by unthreading the jackscrew from the bite block, the screw being turned like a capstan by means of a wire or probe inserted into the transverse holes in the end of the screw (Fig. 2).

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It is obvious that a bite block can be employed for both the maxillary and mandibular arches, and that by using jackscrews on both sides of the mouth any ratio of forces can be applied downward along both mandibular rami simultaneously.

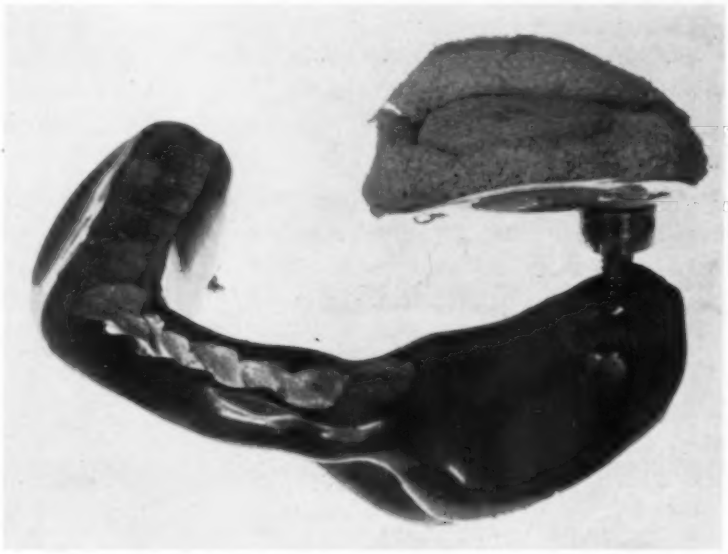

Fig. 1.—Photograph of one form of the appliance used for a patient lacking upper and lower posterior teeth.

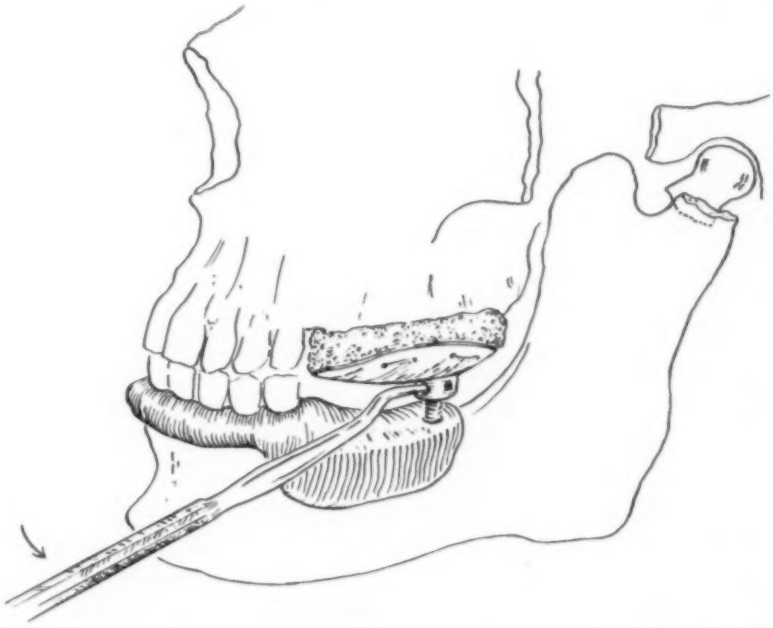

Fig. 2.—Schematic drawing of one form of the appliance used for a patient lacking posterior upper and lower teeth, showing the appliance in place in the mouth and its various parts.

With this appliance satisfactory functional use of the jaw after ten days has been obtained in both uncomplicated collum fractures of the mandible and those associated with fractures of other parts of the bone. In patients lacking posterior molar teeth and having collum fractures, after free motion of the

mandible has been obtained, full occlusion must be maintained by dentures which give tight or balanced occlusion posteriorly. This prevents shortening of the ramus with possible loss of occlusion anteriorly from a downward and backward pull, facial asymmetry, and deviation of the mandible. If dentures are not immediately available the bite block itself can be used temporarily after the jackscrew has been removed. In collum fractures associated with fractures elsewhere in the mandible immobilization of the fracture fragments of the body or ramus of the mandible by external skeletal fixation with the Haynes apparatus[2-5] permits earlier treatment of the collum fracture and mobilization of the jaw.

The described appliance for exerting continual force vertically downward along the line of the mandibular ramus gives satisfactory results with fractures of the coronoid process, in maintaining vertical dimension after arthroplasties on the temporomandibular joints, and with collum fractures of the mandible.

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INTERRELATION OF LARGE PARENTERAL DOSES OF ESTROGEN AND VITAMIN A AND THEIR EFFECT ON THE ORAL MUCOSA

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IN VITAMIN A deficiency certain specific pathologic changes have been observed in numerous epithelial structures throughout the body. Wolbach and Howe[1] have seen a keratinizing metaplasia appearing earliest and most often in the mucous membranes of the trachea and bronchi. They also observed it in the conjunctiva, cornea, accessory sinuses, salivary glands, ureter, uterus, and periurethral glands. Other investigators have reported that excessive cornification of the vagina of the albino rat is an early manifestation of vitamin A deficiency.[2, 3]

King[4] fed rats on a vitamin A and carotene deficient diet for a long period of time (from weaning for varying periods up to fifty-five weeks). The hyperplastic epithelium in the gingival and subgingival tissues of these animals showed a thickened keratinized outer layer and sometimes, finger-like projections into the underlying connective tissue. Similar changes were produced by Mellanby[5] in dogs.

According to the observations of Wolbach and Howe,[6] the fundamental lack of vitamin A appears to involve an atrophy of the epithelium accompanied by or followed by a reparative proliferation of the basal cells. The latter, having lost their type specificity, produce a stratified keratinizing epithelium regardless of the type previously existing in that location (Plate I, Figs. *A, B, C*). On recovery, each epithelium returns to its normal type, indicating no serious morphologic change. The changes in both man and animals are essentially the same.[7]

More recently, studies demonstrating hyperkeratinization of the gingivae and oral mucous membranes resulting from long-term estrogenic therapy have been reported by Ziskin, Blackberg and Slanetz,[8] and Ziskin.[9, 10] They stated that the response to estrogenic hormonal therapy as given was unusually rapid basal cell activity and cell specialization, resulting in the production of keratin not only in the usual areas, but also in such unusual areas as the stratum germinativum and on the surface in the areolar mucosa and cheek mucous membranes.[8]

The reactions of the accessory female genital organs to exogenous or endogenous estrogens are well demonstrated by a study of histologic sections at intervals after administration of the hormone to castrate test animals.[11] The striking transformation of the vaginal epithelium into a hyperkeratinized structure affords a good test for the estrogenic hormone. As the effect of the hormone wears off, the cornified layers are desquamated into the lumen and the vaginal wall returns to its normal state.[12]

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The estrogenic hormone has been found useful in the treatment of vaginal gonococcal infections in children by "maturing" the epithelium. The keratinization thus produced was first reported by Lewis[13] and later confirmed by others.[14, 15]

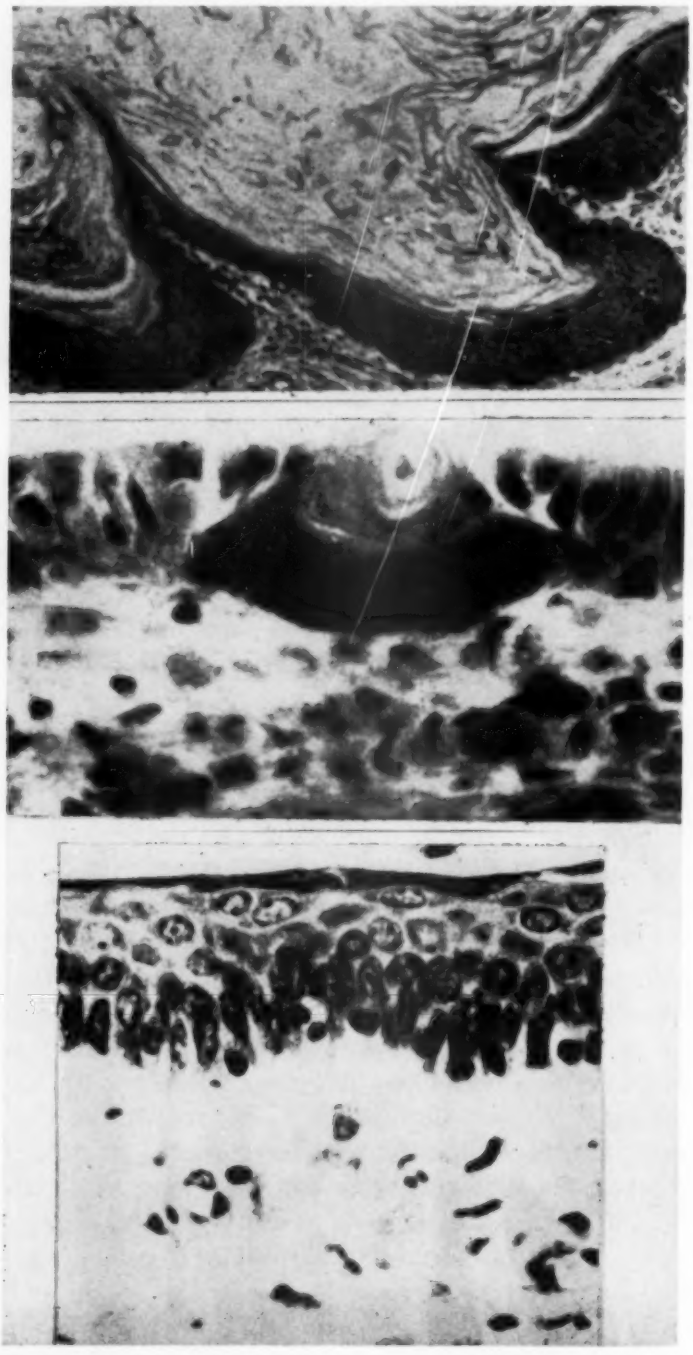

Plate I.—Republished by permission of Dr. S. B. Wolbach. *A* and *B*[6] appeared in Plate 39, Figs. 10 and 12. *C*[1] appeared as Fig. 7. These illustrations show transformation to stratified squamous keratinized epithelium after feeding vitamin A-deficient diets. *A*, Bladder of rat. (×95.) *B*, Respiratory mucosa of rat. (×656.) Early focus of keratinization. *A* and *B*, Sectioned on 181st day of A-deficient diet. Initial age 175 days. *C*, Section of trachea of guinea pig. (×700.) Shows complete replacement by stratified keratinizing epithelium. Fed 110 days on A-deficient diet. Initial age 122 days. (From Arch Path. 5: 239, 1928.)

These studies demonstrate conclusively that hormones and vitamins have specific effects on the organism as a whole. Since both substances individually may affect epithelial structures in a definite manner, the question arises: is their action of an independent nature without regard to the absence or presence of the other, or does some relationship exist?

Attempts have been made at solving this problem by observations on the vaginal mucosa of various experimental animals.

Burrill and Greene[16] reported that the administration to rats of relatively large doses of vitamin A (5,000 U.S.P. units daily for thirty days) produced no effect on the normal estrous cycle, nor did it modify the action of the estrogens on the vaginal epithelium of castrate rats. These findings are in opposition to those of Sherwood, Brend, and Roper,[17] who found that feeding rats carotene (1,500 I.U. for fifteen days to one group and 3,750 I.U. to another) suppressed the normal estrous cycle. Brody and Goldman[18] used castrate rats kept in estrous with minimal doses of estrogen. Daily doses of vitamin A (10,000 to 40,000 I.U. in sesame oil) given simultaneously for two weeks had no effect upon the vaginal smears. Normal animals whose estrous cycle was previously determined were fed daily doses of vitamin A (2,000 I.U. in 2 c.c. sesame oil) for three weeks without any apparent effect on the normal estrous cycle.

The disagreement here cited may be the result of the different products used, the varying lengths of time during which experiments were conducted, and the variation in dosage. The observations of Pincus and Werthessen[19] indicate that estrogenic administration over a short time (five to ten days) leads to thyroid enlargement, whereas injections over a longer period (twenty days or more), lead to thyroid involution. Such findings suggest that thyroid atrophy or depression may play a part in the etiology of hyperplasia and hyperkeratinization of epithelium by altering tissue metabolism.

The present report deals with two experiments designed to test a possible relationship between vitamin A and the estrogenic hormone.*

The first experiment extended through a period of ninety-two days. Daily intramuscular doses of estrogenic hormone were given for a certain period; then vitamin A was added to the estrogenic hormone, and the combined solution was administered daily.

In the second experiment, extending over a period of sixty-six days, vitamin A was administered daily for a certain period; then the estrogenic hormone was added to the vitamin A and the combined solution was given daily.

METHOD

In the first experiment, two subadult female rhesus monkeys, one normal (665) and one castrated (666) were used. The initial weight of each of these animals was 2650 grams. Ovariectomy was performed one week before the introduction of hormonal therapy.

Preexperimental biopsies were made of one of the interdental papillae. Thereafter, daily intramuscular doses of estrogen [progynon B (alpha estradiol benzoate) 1,000 R.U. in 1 c.c. sesame oil] were given for thirty-two days, after

*Experiment 1 reported at meeting of New York Section, International Association for Dental Research, May 28, 1942. Experiment 2 reported at combined meeting of New York Section, International Association for Dental Research, and Dental Section, American Association for the Advancement of Science, Dec. 28, 1942.

(Experiment 2 supported in part by a grant from the Nutrition Research Laboratories, Chicago, Ill.)

which time biopsies were again made. These injections were continued until the fifty-third day, when microscopic examination of the gingivae showed a pronounced estrogenic effect. At this point the dosage was reduced to 500 R.U. The administration of vitamin A was begun on the sixty-first day. Each animal received a daily intramuscular injection of 10,000 I.U. vitamin A (in corn oil) plus 500 R.U. progynon B for thirty-one days, after which time a single injection of colchicine (1 mg. per kilo body weight) was given about eight hours before death.

The animals were weighed and examined at weekly intervals. Their diet consisted of bananas, oranges, whole wheat, carrots, dog biscuit, raw potatoes, hearts of lettuce, egg, rice, and raisins and was divided generally into about 150 to 155 grams of carbohydrate, 14 to 27 grams of protein and between 15 and 20 grams of fat a day.

Post-mortem material was obtained by resecting most of the gingivae, mucous membranes, and vagina, immediately after the animals were sacrificed. The tissues were fixed in Bouin's solution and embedded in paraffin. Serial sections were stained, some with hematoxylin and eosin; others with Masson's trichrome stain.

In the second experiment one subadult normal female rhesus monkey (702) was used. Initial weight was 4,300 grams. Pre-experimental biopsy was made of one of the interdental papilla. Thereafter, daily intramuscular injections were given of vitamin A (10,000 I.U. in 1 c.c. sesame oil) for one month.

Following this, a biopsy was made of an interdental papilla on the other side of the mouth. Thereafter, daily intramuscular injections of combined vitamin A (10,000 I.U.) and progynon B (1,000 R.U.) were given for thirty-six days, following which biopsies of other interdental papillae and one cheek were made. Diet and histologic technique were similar to those in Experiment 1.

RESULTS OF ESTROGEN THERAPY

EXPERIMENT 1.—With the administration of estrogen alone (progynon B), the usual gross changes were seen in both animals in ten days. These were a pronounced edema and wrinkling of the skin around the vagina, which gradually spread over the sexskin and included the back and face; and an accompanying loss of hair over these areas.

Monkey 665 showed a maximum weight increase to 3,160 grams after twenty-five days of estrogenic treatment. Monkey 666 (castrate) showed a weight increase to 3,350 grams.

The gross gingival changes were slight thickening and blanching of the tissues.

Microscopic Findings.—The most noteworthy action of the estrogenic hormone on the oral tissues was the production of a hyperkeratinization on the gingivae as well as a keratinization of the mucous membranes, normally nonkeratinized (Plate II, Fig. 2; Plate III, Fig. 4).

Alveolar Gingivae: Hyperplasia of the epithelium and reduction or abolition of subepithelial inflammatory infiltration were found. In addition, "pearls" were seen in the prickle cell layer. There was proliferation of the basal cells into the corium. The cells appeared crowded with an increase in the number of mitotic figures. The epithelial pegs were pointed, split more frequently than normal, and extended further into the corium. The corium also

showed changes indicative of hyperplasia. The connective tissue was denser and showed an increased number of young fibroblasts and capillaries.

Areolar Gingivae: While normally there is no keratin in this area, microscopic examination showed a parakeratosis three to five cells deep and a definite layer of keratin. Throughout portions of the surface there were seen "epithelial curls."

Changes in the corium were similar to those noted for the alveolar gingiva corium. The tissue was cellular beyond normal.

The foregoing gross and microscopic changes are similar to those previously reported in connection with other studies.

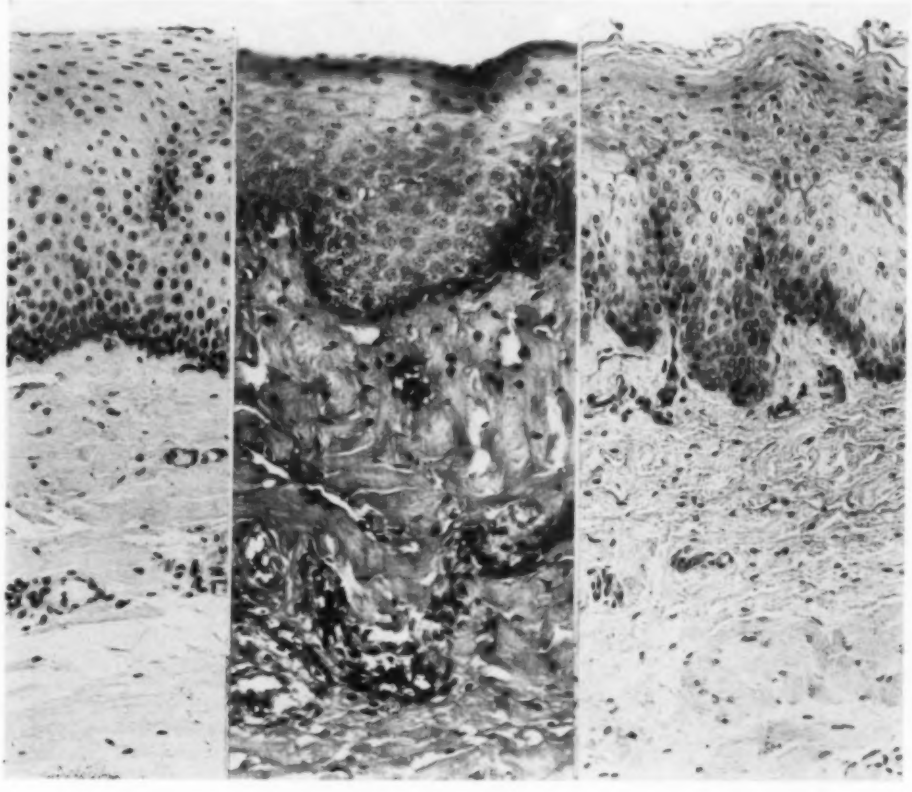

Fig. 1. Fig. 2. Fig. 3.

Plate II.—Monkey 665. Normal female rhesus monkey. Experiment 1. Fig. 1, Pre-experimental section of areolar gingiva. (×150.) No treatment given. Fig. 2. Same animal as in Fig. 1, after fifty-three days of daily intramuscular injections of 1,000 R.U. of progynon B. Areolar gingiva. (×160.) Shows marked hyperplasia of epithelium and connective tissue. Keratin is seen on surface where no keratin exists normally. Note also increased density of connective tissue. Fig. 3, Same animal as in Figs. 1 and 2. After thirty-one days of daily intramuscular injections of combined progynon B (500 R.U.) and vitamin A (10,000 I.U.) following the fifty-three-day period of progynon B injections. Total dosage was 72,500 R.U. progynon B and 31,000 I.U. vitamin A. Areolar gingiva. (×157.) Shows amelioration effect of the vitamin A. There is a regression of the keratinization and of the hyperplasia in both epithelium and connective tissue. There is a tendency to return to the pre-experimental state. Fig. 3 more closely resembles Fig. 1 than it does Fig. 2.

RESULTS OF HIGH VITAMIN A THERAPY

Experiment 2.—Grossly, there were no changes observed. Biopsy made after administration of vitamin A revealed several definite changes in the tissues when compared with pre-experimental biopsy.

Alveolar Gingivae: There was slightly less keratin present. Also the keratin was changed in quality, being more easily fragmented and separated

from the mass. The entire epithelial layer was thinner than formerly, the pegs remaining full and well rounded. The basal cells were reduced in number, and were in more regular even alignment. There was slight intercellular edema, and the intercellular bridges appeared more definite, wider, and deeper staining. This was seen in most of the prickle cell area, extending down to the basal cell layer. There was also a greater number (almost doubled) of mitotic figures. The corium showed increased vascularity and edema. Also, the connective tissue seemed to be present in smaller bunches, as if more easily fragmented. Many more fibroblasts were present than fibrocytes. (Plate V, Figs. 10 and 11).

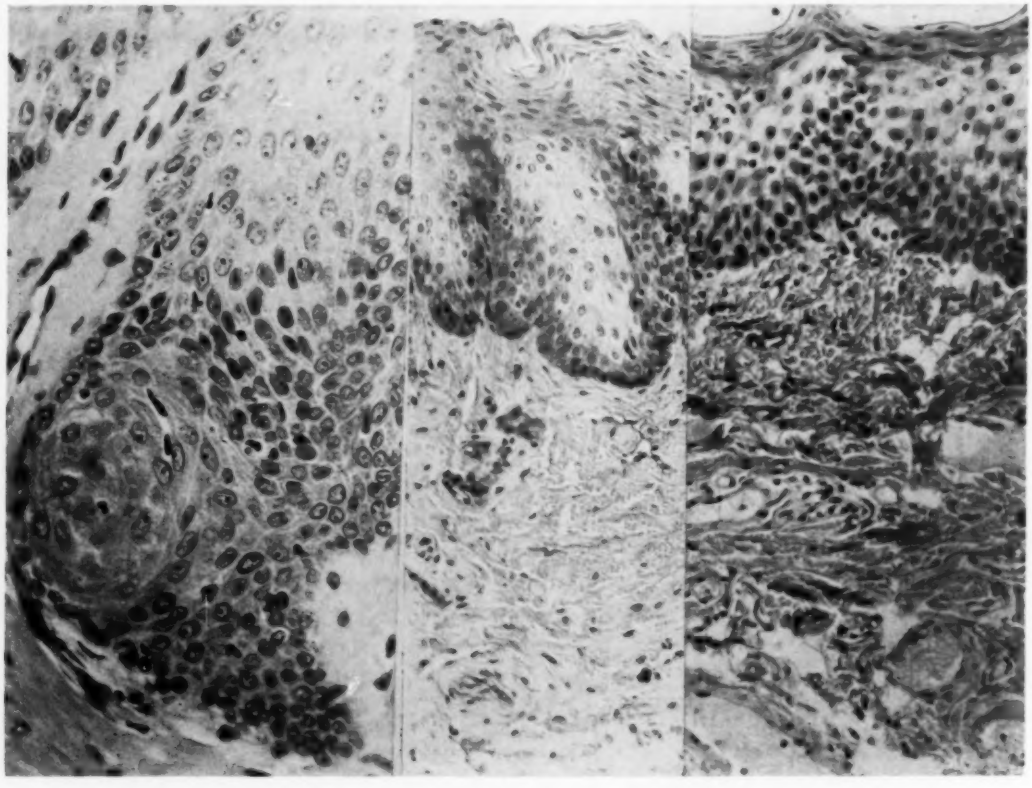

Fig. 4. Fig. 5. Fig. 6.

Plate III.—Fig. 4, Monkey 665. Same animal as shown in Plate II. Alveolar gingiva. (×307.) 1,000 R.U. progynon B injected daily for thirty-two days. Shows hyperplasia of epithelium by increased nuclei in basal cell layer, downgrowth and pointing of epithelial pegs, and pearl formation. Fig. 5, Same tissue as in Plate II, Fig. 3, repeated for comparison with Fig. 6. Fig. 6, Monkey 617. Female rhesus monkey. This illustration is included to show the amelioration effect of both progesterone and time after treatment with estrogen. This animal was ovariectomized, then was given 10,800 I.U. progynon B in seventy-four days. After a period of no treatment for six months, 1,400 mg. progesterone were injected in fifty-six days. Thereafter, no further treatment was given for two years. At this time, this section of tissue was taken. Areolar gingiva. (×170.) Shows regression from estrogenic effect but no resemblance to tissues from untreated ovariectomized monkeys previously reported (J. Dent. Res. 19: 385, 1940: Fig. 3). More closely resembles tissue in Fig. 5.

Areolar Gingivae: Stripping was prominently present at the uppermost epithelial layers. There were intercellular edema and fewer basal cells. Increased vascularity in the corium and edema were present, along with a greater number of fibroblasts than fibrocytes (Plate VI, Figs. 14 and 15). Some of these changes are similar to those reported by Ziskin and Stein,[32] as occurring in oral mucous membranes of monkeys and humans in hypothyroidism. An increased amount of mucinous material taking the carmi-mucin stain was seen.

RESULTS OF COMBINED ESTROGEN AND VITAMIN A

EXPERIMENT 1.—The weight of both animals returned to pre-experimental levels following the administration of vitamin A in conjunction with the estrogen. The sexskin change was not nullified. The most striking microscopic changes were seen in the areolar gingivae where there was a reduction of the epithelial hyperplasia and a pronounced amelioration of keratin formation. There was no longer a heavy definite layer of surface keratin. The cells showed a regressive tendency with many pyknotic nuclei at the surface. This tissue more closely resembled the pre-experimental section. In the alveolar gingivae the layer of keratin was not noticeably lessened, but the epithelial hyperplasia was reduced and there was no evidence of pearl formation (Plate II, Fig. 3; Plate III).

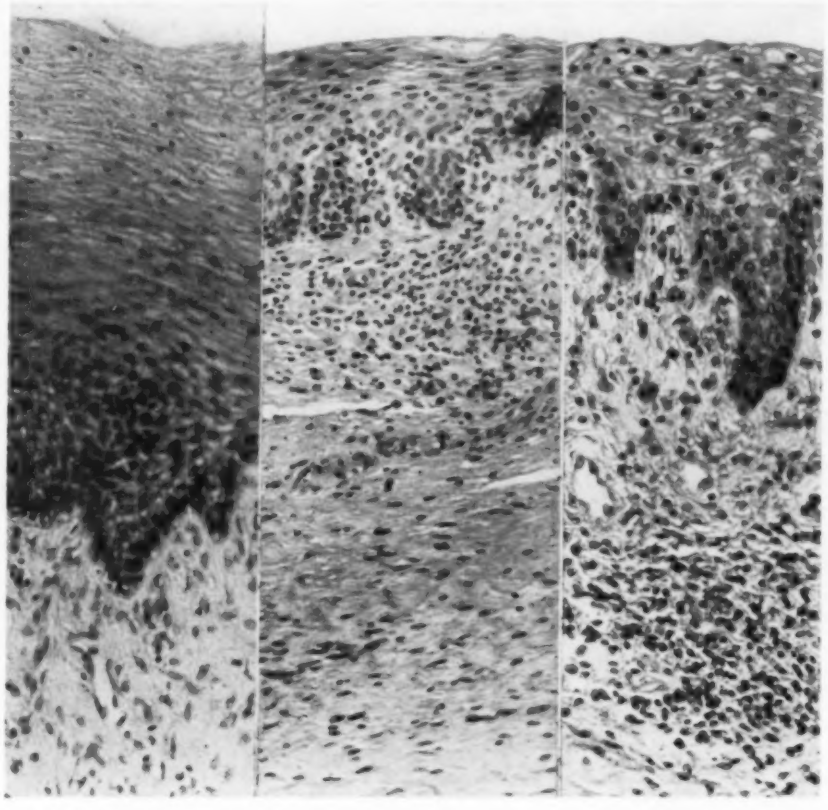

Fig. 7. Fig. 8. Fig. 9.

Plate IV.—Fig. 7, Monkey 452. Female rhesus monkey, ovariectomized. After a period of no treatment for three months, 315,000 I.U. progynon B were injected in 2½ months. Section from vagina. (×157.) Shows hyperplasia and hyperkeratinization of epithelium. Fig. 8, Monkey 605. Female rhesus monkey. Normal. No treatment. Section from vagina. (×157.) To be compared with Figs. 7 and 9. Fig. 9, Monkey 666—Experiment 1. Female rhesus monkey. Ovariectomized and treated with 72,500 R.U. progynon B in ninety-two days. During the last thirty-one days, 310,000 I.U. vitamin A were injected in conjunction with the progynon B. Section from vagina. (×180.) Shows tendency to return to normal state and demonstrates amelioration by vitamin A on estrogen effect. Note reappearance of inflammatory exudate in corium which is absent in Fig. 7.

Post-mortem sections of cheek mucous membranes showed changes similar to those seen in the areolar gingivae.

In studying the influence of vitamin A and estrogens on tissues other than the gingivae, changes resembling those found in the mucous membranes were ob-

served in the vaginas of both animals. The most marked feature was a reduction in the amount of keratinization of the surface epithelium after the administration of vitamin A (Plate IV, Fig. 9).

EXPERIMENT 2.—Grossly, there was immediate enlargement and reddening of the sexskin with slight loss of hair. The redness extended well down the back of the thigh and remained throughout the term of estrogen administration. No gross gingival change was evident. There was no significant weight change throughout the experiment.

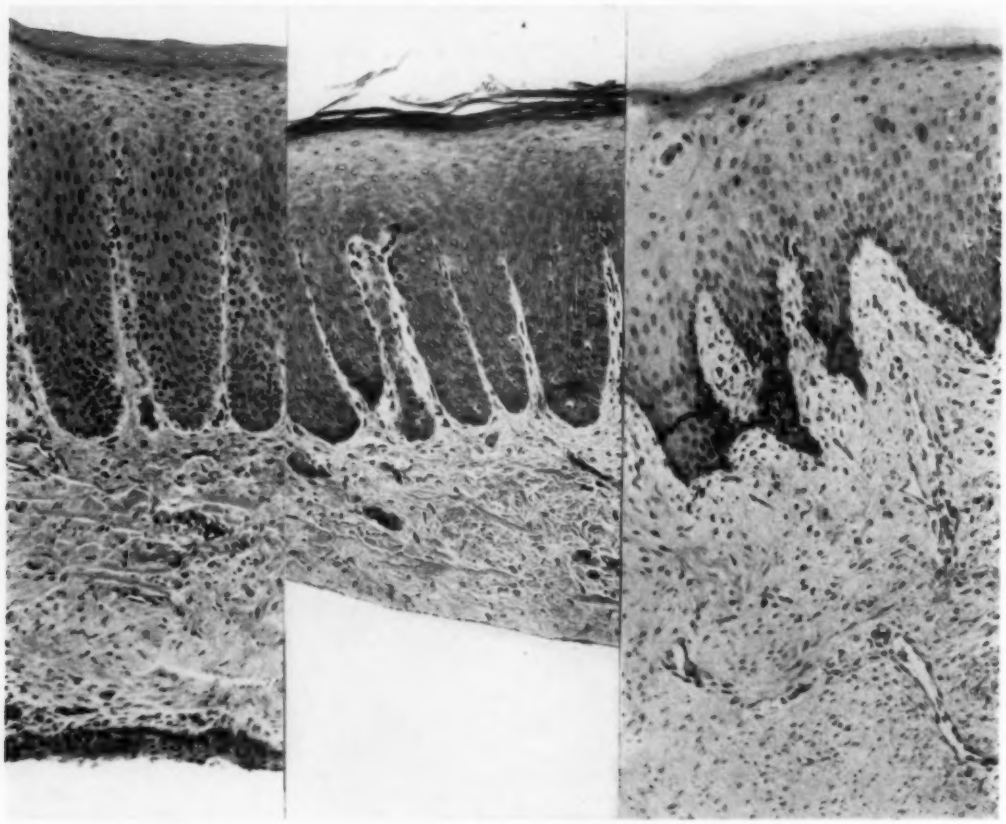

Fig. 10. Fig. 11. Fig. 12.

Plate V.—Monkey 702. Normal female rhesus monkey. Experiment 2. Fig. 10, Pre-experimental section of alveolar gingiva. (×98.) No treatment given. Shows normally rounded epithelial pegs and a definite keratin layer. The connective tissue also appears firm with no inflammatory reaction seen. Fig. 11, Same animal as in Fig. 10. After thirty days of daily intramuscular injections of 10,000 I.U. vitamin A. Alveolar gingiva. (×98.) Shows tissue degeneration evidenced by fewer cells in basal layer, keratin layer reduced in thickness, not as uniform as the normal, and becomes fragmented easily in sectioning. Epithelial peg pattern is not altered greatly, but both the epithelium and connective tissue are reduced in thickness. Fig. 12, Same animal as in Figs. 10 and 11. After thirty-six days of combined vitamin A (10,000 I.U.) and progynon B (1,000 R.U.) injected daily. Alveolar gingiva. (×98.) Shows hyperplasia of both epithelium and connective tissue. The epithelial hyperplasia is evidenced by increased number of nuclei in basal layer. The change in the peg pattern to pointing is caused by the proliferation, and the hyperkeratinization is evidenced by formation of "pearls," parakeratosis and a more mature keratin layer on the surface. The hyperplasia in the connective tissue is evidenced by increased thickness of this layer. Although taken at the same magnification as Figs. 10 and 11, we were not able to get the entire layer on this photograph. These changes are characteristic of the estrogenic hormone effect showing an amelioration.

Alveolar Gingivae: Microscopically, there were several definite changes. The keratin layer was thicker and extended well over the crest of the papilla. The keratin was intact, no evidence of fragmentation and separation being

present. Epithelial pegs were more irregular in length and more pointing and splitting were seen. Intercellular bridges and intercellular edema were less marked than in the vitamin A sections. Epithelial pearls and "curls" were seen in the alveolar portion. Fewer mitotic figures were seen although several were present. The basal cells were more numerous and crowded; many small ones were present. These changes in the epithelium are indicative of hyperplasia and hyperkeratinization. An increased number of fibrocytes and heavy bundles of collagen fibers were seen in the corium, as well as an increase in thickness, indicating hyperplasia (Plate V, Fig. 12).

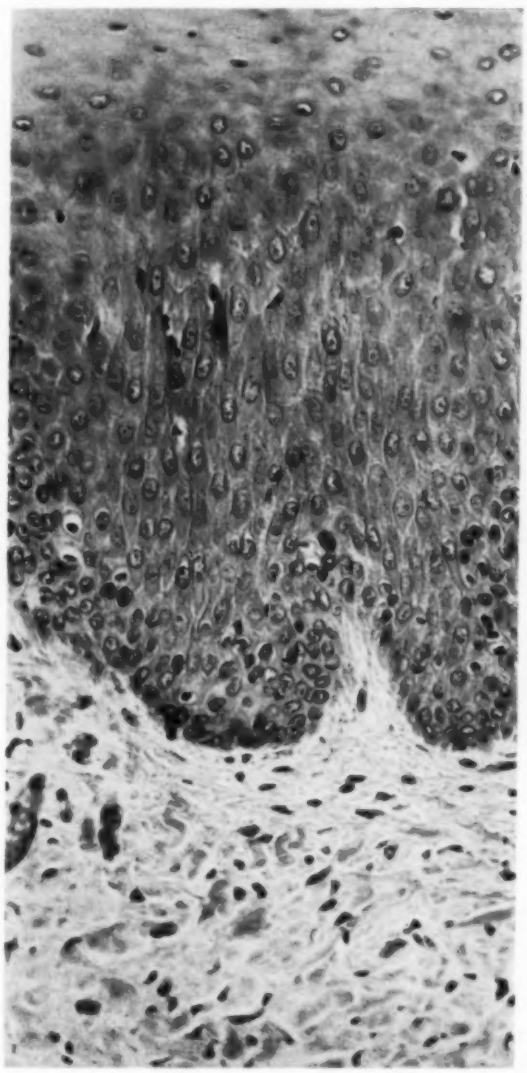

Fig. 13.—High-power view of Fig. 11. (×293.) Shows increased mitosis, prominent intercellular bridges, slight intercellular edema, presence of young connective tissue cells in corium.

Areolar Gingivae: Definite parakeratosis was present, in some areas extending as deep as 5 to 8 cells. "Epithelial curls" were also prominent. Similar changes were seen in the areolar corium as were noted in the alveolar portion, although the structure in the areolar was looser (Plate VI, Fig. 16; Fig. 17).

DISCUSSION

Vitamin A Deficiency Effects.—Several investigators have reported that an early manifestation of vitamin A deficiency is continuous cornification of the vaginal epithelium of the albino rat. Evans and Bishop[20] found such changes in the intact and castrate rat. Evans[21] and Mason[22] reported that in the intact animal this cornification is independent of the estrous cycle and does not indicate a continuous state of estrous because ovulation and corpus luteum formation occur at intervals as shown by successful mating with normal males. Turner and Loew[23] reported similar continuous cornification of the vagina occurring in the A-deficient monkey.

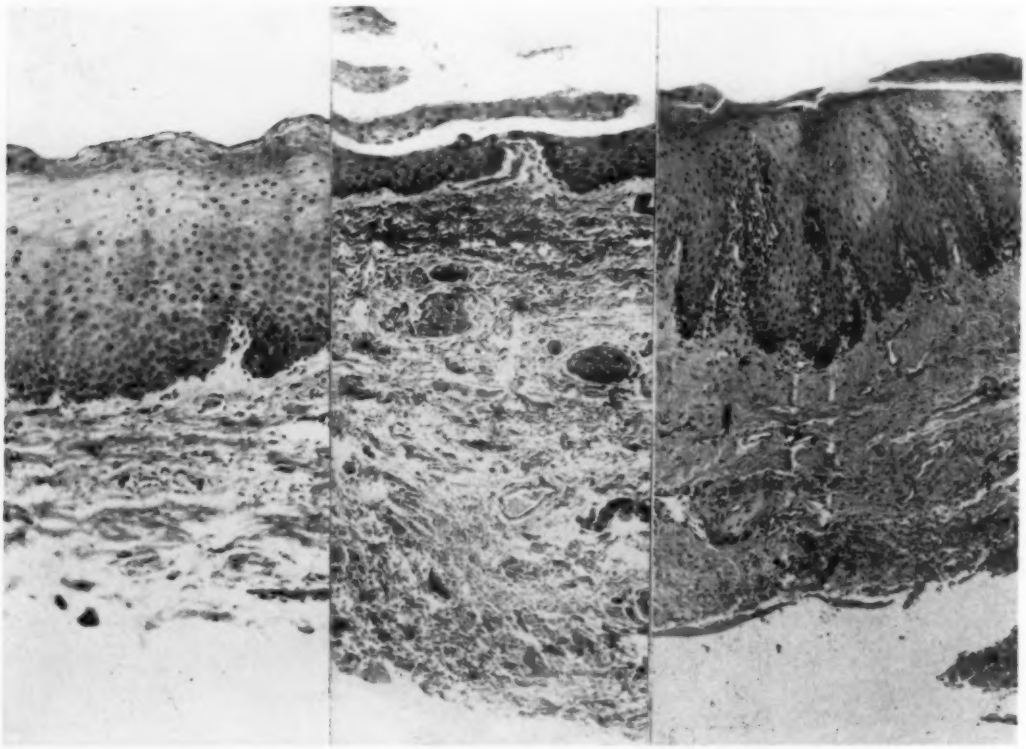

Fig. 14. Fig. 15. Fig. 16.

Plate VI.—Monkey 702. Same animal as shown in Plate V. Fig. 14, Pre-experimental section of areolar gingiva. (×85.) No treatment given. Shows normal structures. Fig. 15, Same animal as in Fig. 14, following thirty days of daily intramuscular injections of 10,000 I.U. vitamin A. Areolar gingiva. (×85.) Shows degenerative changes evidenced by stripping of epithelium and edematous connective tissue. Fig. 16, Same animal as in Figs. 14 and 15, following thirty-six days of daily intramuscular injections of 10,000 I.U. vitamin A and 1,000 R.U. progynon B. Areolar gingiva. (×85.) Shows hyperplasia of epithelium and connective tissue. Note parakeratosis and keratin "curls" on the surface where keratin is not normally seen. Note also increased thickness and solid appearance of connective tissue. Demonstrates amelioration by the estrogen.

Extensive experiments and clinical studies have established the concept that among the various manifestations of vitamin A deficiency are the characteristic metaplasia and keratinization and loss of secretory function in epithelial tissue throughout the body. The vitamin probably acts as a catalyzer, or regulator of metabolic processes, particularly essential to the functional and structural integrity of epithelial cells.

Simpson and Mason[24] reported beneficial effects of vitamin A in the treatment of senile vaginitis in postmenopausal women whose dietary history suggested an insufficient intake or utilization of vitamin A.

Mason and Ellison[25] suggested that vitamin A plays a part in protein metabolism within the epithelial cell. In deprivation of the vitamin, the formation of glyco-protein or mucin seems to be retarded and that of albuminoids or keratin to be increased or appear in excess.

Recently, cutaneous lesions, considered to be the result of vitamin A deficiency, have been reported. Peck, Chargin, and Sabotka[26] cited several cases of dyskeratosis follicularis. These patients, although on a vitamin A containing diet, showed a decided decrease in the vitamin A content of the blood serum, while the carotene content was within normal limits. The disease was successfully treated with large doses of vitamin A (200,000 I.U. orally per day), and the blood level of vitamin A was restored to normal. These writers suggest that the disorder was probably due to a defect either in vitamin A absorption from the gastrointestinal tract or in the conversion of the provitamin into vitamin A which reflected itself in the epithelial structure as a physiologic disturbance. Althausen and Stockholm[27] have stated that absorption from the gastrointestinal tract is regulated by the level of thyroid activity.

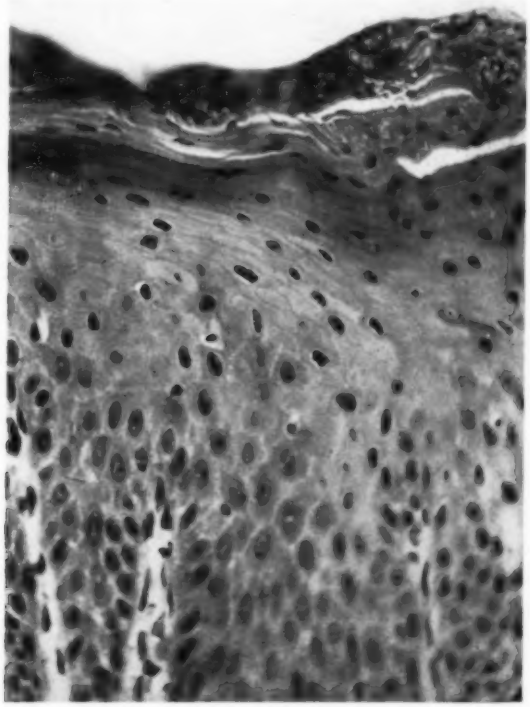

Fig. 17.—High-power view of Fig. 16. (×356.) Shows parakeratosis and keratin "curl."

Thyroid and Vitamin A.—Fasold and Heidemann[28] reported that the milk of goats, which is normally white, is yellowish after thyroidectomy, notwithstanding the fact that a normal diet is maintained. The author attributes the color change to an apparent failure to convert carotene into vitamin A. Several have suggested that the thyroid hormone was essential for the conversion of carotene and also for the storage of vitamin A in the liver.[28, 29, 30, 31]

It has also been observed[29] that the vitamin A in the blood of cretins and of myxedematous patients is of decreased concentration or absent, while the value for carotene usually is high. This vitamin A deficiency in cretins and in myxedematous patients is given as a possible reason for the texture of their skin and their pathologic adaptation to darkness. Striking general improvement of patients who had hypothyroidism was reported following administration of diets rich in vitamin A.

Ziskin and Stein[32] state that in experimental hypothyroid monkeys and in cretins there is produced a hyperkeratinization of the oral mucous membranes in some respects resembling that seen in vitamin A deficiency and long-term estrogenic therapy.

Reuter[33] found histologic changes in the skin of cretins and myxedematous individuals similar to those observed by Ziskin and Stein in the oral mucous membranes.

The histologic changes noted in our experiments following administration of high doses of vitamin A which were similar to those seen in the hypothyroid state would suggest that there may be an antagonistic action of hypervitaminosis A on the thyroid. However, other experimental work on rats cited in published reports is not conclusive. The work of Euler and Klussmann,[34] Rappai and Rosenfeld,[35] and Abelin[36] suggested such antagonistic action. That this action may take place through the effect of vitamin A on the thyrotropic principle of the pituitary is indicated by the results of Schneider,[37] and Fellinger and Hochstädt.[38] Using guinea pigs, they found that the histologic changes which the thyrotropic principle of the pituitary usually caused did not appear when large amounts of vitamin A were given. On the other hand, the findings of Moore,[39] Wolff,[40] and Fasold and Peters[41] would appear to deny the existence of this antagonistic action. Baumann and Moore,[42] in a series of experiments on rats in which they gave toxic but not rapidly fatal doses of vitamin A and thyroxin, stated that the action was additive rather than antagonistic. These varying reports indicate the need for further study along these lines, especially as they relate to the dosages used in the experiments.

Thyroid and Estrogens.—Many studies have been undertaken on changes in metabolism during various stages of the estrous or menstrual cycle. Sherwood and Bowers[43] reported that the basal metabolism of hyperthyroid rats decreased 10 to 54 per cent following injections of estrogen and returned to normal when 100 R.U. were injected for three days. Sherwood, Wilson, and Boneta[44] stated that following thyroidectomy, estrogen had no influence on the metabolic rate. Gessler[45] found that a decrease in basal metabolism of normal and hyperthyroid guinea pigs followed injections of estrogen. Danforth, Green, and Ivy[46] reported that estrogen had little effect on metabolism of normal rats but prevented, in part, the effect of thyroid when the two hormones were given together.

Estrogens and Metabolism of Specific Tissues.—It has been noted that as estrogens produce profound changes in specific tissues they may alter the metabolic activity of certain parts of the body and not others. Khayyal and Scott[47] and King[48] found an increased oxygen consumption of the uterus of rats preceding ovulation. David[49] and Aschheim and Gesenius[50] found that the metabolism of the uterus also increased following the injection of estrogen.

Cyclic changes in both aerobic and anaerobic glycolysis were observed in the rat by Kerly.[51] Khayyal and Scott[52] saw an increased oxygen consumption of the uterus following the injection of follicular fluid. An increased oxygen consumption of pituitaries in rats was noted during proestrum and estrous by Victor, Anderson, and Prest.[53] It was at lowest level during dioestrum and in castrated rats.

Diabetes and Vitamin A.—Ziskin and Siegel[54] reported hyperkeratinization in the oral mucosa of diabetic children. Generally, these cases were found to have a higher blood carotene and lower blood vitamin A content than normal children.

Vitamin A Deficiency and Estrogen.—No attempt has been made to determine whether the keratinizing process of vitamin A deficiency differs in any respect from that normally occurring under the influence of hormonal factors. It is an established fact that the estrogens alone are capable of influencing keratin formation. However, our problem concerns itself with a consideration of the estrogens when given in sufficiently large doses and over a long enough period of time to make plausible the hypothesis that vitamin A has, by these means, been depleted. Under these conditions the theory may be advanced that the estrogenic hormone induces keratinization and hyperplasia in a manner resembling vitamin A deficiency in epithelial structures. The mechanism here involved may be explained in one of three ways:

1. The estrogenic hormone may suppress the anterior pituitary which in turn depresses the thyroid gland. This lack of thyroid secretion, since it appears essential for the conversion of provitamin A into vitamin A, may be responsible for the changes in the epithelial tissue.

2. The estrogens may directly suppress thyroid activity and metabolism in general, thereby causing failure of conversion of provitamin A into vitamin A. In addition, the metabolism of the epithelial tissue is thus reduced with a consequent decrease in the utilization of vitamin A.

3. Estrogens may exert a direct effect on oral epithelial tissues, increasing their metabolism so that the vitamin A, which is believed to be a regulator of cellular integrity, is rapidly depleted causing the subsequent morphologic changes to occur.

If any of these theories is applicable, the replacement of the deficient vitamin A after estrogenic therapy should cause a regression or suppression of the keratinization and the tissues should tend to become more normal in character.

Our findings in Experiment 1 indicate the possibility of such a relationship in that, upon injection of vitamin A, the estrogenic effect was nullified to some degree.

SUMMARY

The problem is one of potential connection between the hyperkeratinization of oral mucous membranes seen in vitamin A deficiency and that produced in long-term large-dose estrogenic therapy. Hormones and vitamins have specific effects on the organism as a whole. Both of these substances affect epithelial

structures in a definite manner. Do they at all times perform as separate entities or, under the conditions described, do they act, directly or indirectly, upon each other?

In the first experiment, two subadult female rhesus monkeys, one normal and one ovariectomized, were given 1,000 R.U. of progynon B for fifty-three days, when biopsies showed a definite change in the alveolar and areolar gingivae as evidenced by hyperkeratinization and hyperplasia. Eight days after the observance of these changes, daily doses of vitamin A (10,000 I.U.), plus estrogen (500 R.U.), were given for thirty-one days, after which time the animals were sacrificed and sections of oral mucous membranes and vaginas were studied. The effect of the vitamin A upon the keratinization and hyperplasia produced by estrogen was a suppression or amelioration.

In the second experiment on a female rhesus monkey, large doses of vitamin A (10,000 I.U.) daily for thirty days were followed by combined vitamin A (10,000 I.U.) and estrogen (1,000 R.U.) daily for thirty days. In general, the results of the vitamin A administration indicated a degenerative change in the gingivae. The addition of estrogenic hormone to the administration of vitamin A resulted in overcoming the high vitamin A effect, producing hyperplasia of the gingivae with marked restoration of tissue tone. Although the injection of the combined estrogen and vitamin A produced an amelioration of the previous treatment effects in both experiments, the results in each were dissimilar.

Acknowledgment is made to the Schering Corporation of Bloomfield, N. J., for their courtesy in supplying the progynon B used in these experiments; and to Drs. Schwenk and Gilbert for their cooperation.

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Case Reports

CASE NO. 77

GLOBULO-MAXILLARY CYST

H. J. Field, D.D.S., and A. A. Ackerman, D.D.S.
Newark, N. J.

THE chief complaint was a bad-tasting discharge from the anterior region of the palate.

Clinical Examination.—The past history of this woman, 32 years of age, was irrelevant. Examination of the mouth revealed a normal complement of teeth (Fig. 1). On the lingual aspect of the palate between the upper left lateral incisor and cuspid a depressed area was observed (Fig. 2). A probe was readily passed into this area. There was no tenderness or swelling in the region. The cuspid and lateral incisor were clinically normal and responded normally to the electric pulp tester.

Radiographic Report.—A large radiolucent area was observed between the upper left cuspid and lateral incisor (Fig. 3). At first glance this area suggested a typical radicular cyst. Closer examination, however, revealed a normal peridental membrane and lamina dura around both adjoining teeth. The diagnosis of radicular cyst was therefore untenable. Another abnormal feature was the evidence of loss of cemental structure in the apical regions of both the central and lateral incisors.

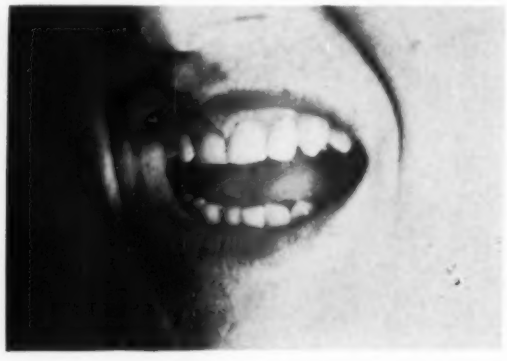

Fig. 1.

Operation.—Following the reflection of the palatal mucosa, a cyst membrane was readily enucleated. A distinguishing characteristic of the membrane was its thickness. The usual dental cyst has a thin, friable wall while in this case the wall was coarse and thick. Sections of the roots of the central and lateral incisors were clearly seen and the absorbed areas of cementum on each

of these teeth were easily visualized. This absorption was obviously caused by pressure from the cyst wall. The apices of neither the lateral incisor nor the cuspid were involved in the cystic area.

Diagnosis.—On the basis of the radiographic and clinical findings we offered a tentative diagnosis of fissural cyst. According to Thoma's classification the proper name for this type of cyst is the globulo-maxillary cyst. Thoma reports a similar case in his *Oral Pathology.*

Pathologist's Report.—Gross: The specimen consists of a flat membranous piece of yellow and white fibrous tissue 2 by 1.5 by 0.3 cm. Both surfaces are ragged and bear coarse nodular projections 1 to 3 mm. in diameter.

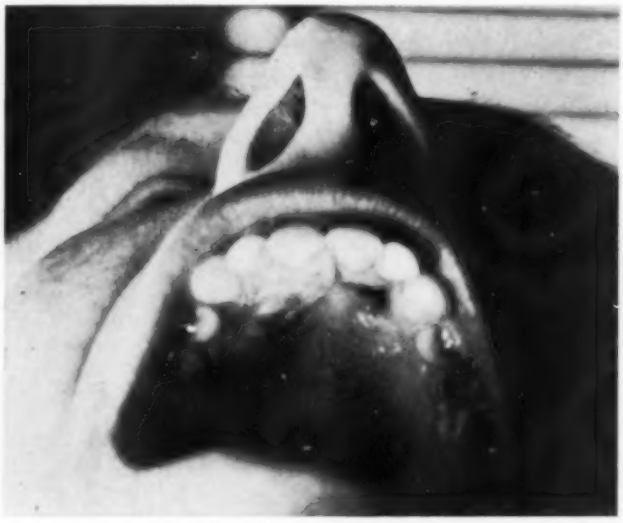

Fig. 2.

Fig. 3.

Microscopic: Sections of the cyst sac wall show it is composed of interlacing dense collagen fibers containing scattered nests of lymphocytes and monocytes. The sac wall is lined with stratified squamous epithelium of variable thickness which sends long slender processes deep into the underlying connective tissue. The connective tissue is well vascularized. The findings are consistent with the diagnosis of fissural or globulo-maxillary cyst.

Diagnosis.—Globulo-maxillary cyst.

CASE NO. 78

NONPIGMENTED NEVUS ON LABIAL MUCOSA

H. J. Field, D.D.S., and A. A. Ackerman, D.D.S.
Newark, N. J.

MISS F. S., aged 35, presented herself with a chief complaint of "small mass on the gum in the upper left anterior region."

Clinical Examination.—The condition of the teeth and supporting soft tissues was normal. The upper left lateral incisor was absent. A bridge extended from the central to the cuspid replacing the missing lateral. On the labial mucous membrane in the region of the extracted lateral incisor a small mass of tissue was observed. This tumor tissue was grayish pink in color and suggested a cluster of tiny eggs. The mass was not movable and was not attached to a pedicle (Fig. 1). It was a painless lesion. The size of the tumor mass was approximately 2 by 1 cm. and its shape ovoid.

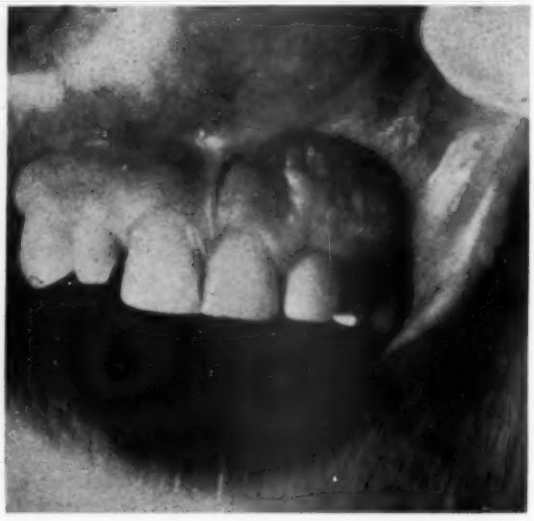

Fig. 1.

Operation.—The entire mass along with several millimeters of normal peripheral mucous membrane was removed with the electrocautery.

Pathologist's Report.—Gross: A cup-shaped piece of gray, firm tissue, measuring 2 cm. in diameter and 3 mm. in thickness, was received.

Microscopic: The slide revealed a section of tissue covered by a moderately acanthotic layer of stratified squamous epithelium. No hyperkeratosis was noted. The underlying connective tissue was rather densely fibrous, moderately vascular, and exhibited some inflammatory exudate in a general perivascular arrangement. In the corium there were sheets of clear round cells rather closely packed

in some areas (Fig. 2). These cells have the appearance of nevus cells, although no melanin pigment can be seen. The appearance of such a lesion in this location is interesting and rather unusual. There is no reason, however, why its occurrence should not be more frequent.

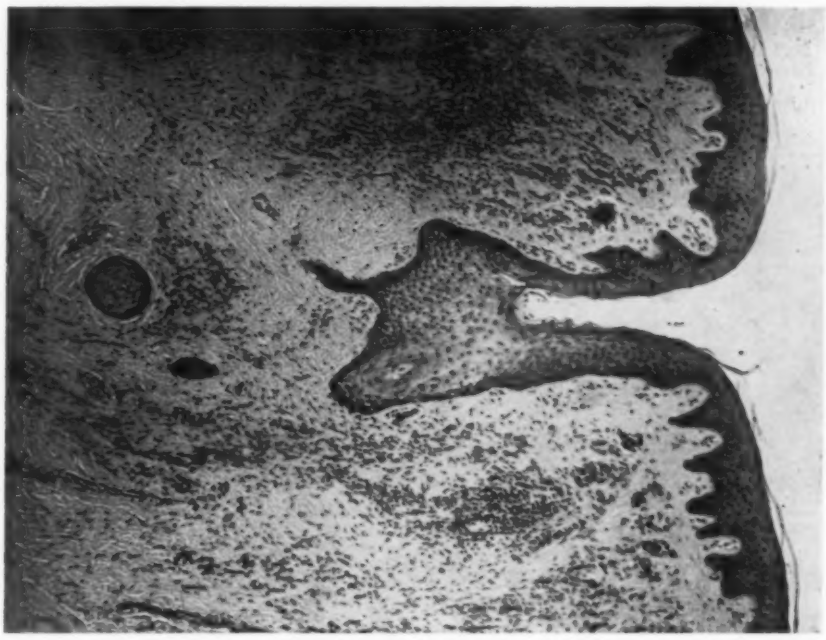

Fig. 2.

Diagnosis.—Nevus, nonpigmented.

Announcement

The annual M. I. Schamberg meeting of the Bronx Hospital Dental Clinic will be held on April 8, 1943, at 9 P.M., at the Hospital, 169th Street and Fulton Avenue. The subject, "The Maxillary Sinus," will be presented by Dr. David Wurzel, Director, Dental Department, Bronx Hospital; Attending Oral Surgeon, Bronx Hospital. Dr. M. I. Schamberg, Attending Oral Surgeon, Bronx Hospital, will be the discussor.

Editorial

External Drainage and Skeletal Fixation in Complicated Fractured Mandibles

One of the most common complications of fractures of the mandible is infection, and infection in turn is often the cause of delayed healing, or nonunion. It is recognized also that proper reduction and fixation of a fractured bone are a fundamental requirement for the healing of the infected wound. The selection of the best method of fixation for a given case is therefore important, and the question of preventing infection and its consequences is a timely topic of discussion.

In many cases of fractures in which union has been delayed, the primary fault in treatment has been ineffective fixation and stabilization of the bone. Generally the surgeon has attempted to treat the case by intermaxillary ligation, though the number or condition of the teeth for such a procedure has been unsatisfactory. These are the cases in which skeletal fixation is useful and should take the place of intermaxillary wiring. Not only does skeletal fixation give excellent results in complicated fractures, but it is also particularly useful if the fracture is associated with extensive wounds of the mouth. It facilitates irrigation and other local therapy of the wound.

External incisions may be used in certain cases either to prevent or to drain abscesses. Whether it should become a routine procedure is under discussion in many places. Rushton,* in a recent article entitled "External Drainage in the Treatment of Fractured Mandibles," discusses the proper use of drainage. His conclusions are based on the experience derived from three years' observation at the Plastic and Jaw Injury Centre E.M.S., Basingstoke, England. He considers that external drainage is indicated: (1) In all cases in which there is an external communicating wound; (2) in all cases in which the fracture hematoma has become destroyed and infected even though no pus be seen or suspected; (3) in cases in which there is a very large hematoma beneath the fracture; (4) in all cases in which there is a collection of pus or a sequestrum at the lower part of the fracture line; and (5) in certain late cases in which there is progressive bone loss.

K. H. T.

*Rushton, Martin A., M.B., L.D.S.: *The British Dental Journal* 73: 283, 1942.


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